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American Journal of Diseases of Children

Vol. 7 JANUARY, 1914 No. 1

LEUKOCYTE COUNTS IN PNEUMONIA AND CEREBRO-SPINAL MENINGITIS *

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CHICAGO

INTRODUCTORY

It has long been recognized that the leukocytes perform an important function in the struggle against bacterial invasion of the organism, and many important contributions have been made to this subject. But very few of these studies have been devoted to infants and young children.

The pneumonias of infants have been particularly slighted, and that fact led to the undertaking of this work, more especially as our clinical experience has caused us to ponder as to the correctness of the assertion that lobar pneumonia was infrequent until after the second year. It is, however, true that most of the cases that come to necropsy under the second year are of the mixed type; these, however, are rarely primary or uncomplicated cases.

To make this work of practical interest, cases of lobar and bronchopneumonia as well as complicated cases have been selected, fifty in all. It was hoped that some information of value for clinical use might be gained when the blood is examined in its relation to the clinical picture in the individual case; first, as to the virulence of the invading strain; second, the reaction on the part of the organism; third, the presence of a true or pseudo crisis, and fourth, the onset of complications.

Interest in the study of the blood in epidemic cerebrospinal meningitis was stimulated by a chance reading of Rusca's article, published some two years ago, on differential blood-counts in a series of nine cases with six deaths and three recoveries, treated without the use of serum. All of his cases were adults, with the exception of one child, aged 5. It was hoped that some information might be gained whereby these children might be saved repeated spinal puncture for diagnostic purposes and unnecessary injections of serum, through the light gained by some of our untoward experiences immediately following such procedures.

Conclusions, so far as they are possible, are based on the absolute counts of the different cells, rather than percentages, the latter being unreliable and misleading in their interpretation, due to the rapid changes in the total counts as influenced by different clinical phases and by treatment.

* Read in the Section on Diseases of Children of the American Medical Association, Minneapolis, June, 1913. This article is abbreviated by omission of case reports. It appears in full in the Transactions of the Section.

A discussion of averages in the various groups of cases has also been avoided because such information is also misleading. Reliable information can only be gained by a careful study of the blood-findings with reference to the clinical manifestations in the individual case.

EHRLICH'S CLASSIFICATION OF LEUKOCYTES

Lymphocytes, large and small.

Size: Small, 5 to 8 microns. Large, 8 to 10 microns.

Number: In adult, 22 to 25 per cent. of leukocytes, 1,500 to 1,700 per cubic millimeter. In infants, 40 to 60 per cent.

Nuclei: Usually central, stain deeply, one or two nucleoli present.

Protoplasm: Usually basophilic, but in old cells sometimes acidophilic.

The larger cells possess an irregular staining nucleus, with a chromatin network and a faintly granular margin of protoplasm. There may be exceedingly large cells in the blood of infants which when stained with the polychrome methylene blue-eosin stains show a distinct granulation in about 50 per cent. of their number.

Large Mononuclear Leukocytes.

Size: Two to three times the size of red-blood cells.

Number: In adult, 1 per cent. of leukocytes, 70 per cubic millimeter.

Nuclei: Large, oval, eccentrically placed, staining much less intensely with the basic dyes than the lymphocytes.

Protoplasm: Weakly basophilic, without granules (Ehrlich stain). Nodal thickenings are present and they show a granulation with the Giemsa and triacid stains.

Transitional Cells.

Size: Resemble large mononuclear leukocytes, but are often larger, being the largest in the blood.

Number: In adults, 1 to 3 per cent. of leukocytes, 70 to 210 per cubic millimeter.

Protoplasm: Stains quite deeply with Ehrlich's stain and often presents a very few neutrophil granules.

Polymorphonuclear Neutrophils.

Size: 10 microns.

Number: In adults, 70 to 72 per cent., 4,900 to 5,040 per cubic millimeter. In infants, 18 to 42 per cent.

Nuclei: Stain deeply.

Protoplasm: Takes a faint acid stain.

Granules: Neutrophilic. Many may be on the nucleus. When migrated these are the ordinary pus cells.

Eosinophils.

Size: The same size or a little larger than the polymorphonuclears.

Number: In adults, 2 to 4 per cent, 140 to 280 per cubic millimeter.

Nuclei: As a rule more faintly staining.

Protoplasm: Slightly more abundant.

Granules: Eosinophilic, but do not lie upon the nucleus.

Mastzellen.

Number: 0.5 per cent., 35 per cubic millimeter.

Nuclei: Polymorphous and faintly staining.

Protoplasm: Acidophilic.

Granules: Basophilic and are not affected by the triple stains.

In the normal differential count there is considerable variation among authorities. This is largely due, however, to the fact that there is no unanimity of opinion as to what types of cells are included in the various groups.

NUMBER OF LEUKOCYTES IN BLOOD-PICTURE IN INFANTS AND CHILDREN

Da Costa gives the following figures for the total and differential leukocyte counts in infancy:

Small lymphocytes, 50 to 75 per cent. First year, 10,000 to 12,000.
Large lymphocytes, 6,000 to 14,000. Sixth year and upward, 7,500.
Polymorphonuclear neutrophils, 28 to 40 per cent.
Eosinophils, 5 to 10 per cent.

TABLE 1.—LEUKOCYTE COUNTS IN 150 HEALTHY INFANTS AND CHILDREN AT DIFFERENT AGES. (D. RABINOWITSCH)

Age, Years	Total Leukocyte Count	Polymorphonuclear Count		Large and Small Lymphocytes		Transitionals		Eosinophils		Mastzellen		Large Mononuclear Neutrophils
		Total	Per Cent.	Total	Per Cent.	Total	Per Cent.	Total	Per Cent.	Total	Per Cent.	
1 to 2.	6,500	1,950	30	3,965	61	162	2.5	234	3.6	25	0.4	
2 to 3.	7,350	2.570	35	4,080	55.5	183	2.5	426	5.8	28	0.4	
3 to 4.	6,400	2,270	35.5	3,360	52.5	192	3	397	6.2	20	0.3	Varies from 1.0 to 3.3 per cent. of total.
4 to 5.	6,560	2,500	38	3,410	52	138	2.1	426	6.5	26	0.4	
5 to 6.	7,100	3,585	50.5	3,020	42.5	192	2.7	213	3	28	0.4	
6 to 7.	6,800	3,370	49.5	2,650	39	184	2.7	367	5.4	34	0.5	
7 to 8.	6,325	3,005	47.5	2,275	36	278	4.4	405	6.4	32	0.5	
8 to 9.	6,140	3,070	50	2,240	36.5	172	2.8	344	5.6	32	0.5	70 to 200
9 to 10.	6,160	3,170	51.5	2,220	36	154	2.5	333	5.4	21	0.3	
10 to 11.	5,850	3,220	55	2,020	34.5	146	2.5	257	4.4	23	0.4	
11 to 12.	6,790	3,600	53	2,340	34.5	163	2.4	510	7.5	34	0.5	
12 to 13.	6,250	3,530	56.5	1,970	31.5	194	3.1	300	4.8	37	0.6	
13 to 14.	6,890	3,995	58	2,135	31	248	3.6	296	4.3	28	0.4	
14 to 15.	7,000	4,585	65.5	1,750	25	217	3.1	266	3.8	19	0.3	
15 to 16.	8,000	5,440	68	1,840	23	256	3.2	328	4.1	50	0.6	

Morse's figures for leukocyte counts in infants under 2 years are as follows:

Total, 10,000 to 14,000.
Polymorphonuclears, 35 to 45 per cent.
Small mononuclears, 40 to 50 per cent.
Large mononuclears, 10 per cent.
Eosinophils, 1 to 5 per cent.

Rabinowitsch (Table 1), from his studies, came to the following conclusions:

1. Healthy children between the ages of 1 and 15 years average between 6,000 and 7,000 leukocytes, approximately the same as adults.

2. Polymorphonuclear neutrophils increase gradually from 30 per cent. in the first year to about 70 per cent. in the fifteenth year.

3. Lymphocytes decrease from 60 per cent. in the first year to about 30 per cent. in the fifteenth year.

4. The reversal of the percentages of neutrophils and lymphocytes occurs usually about the sixth year.

5. Eosinophils average between 4 to 6 per cent., but vary greatly in different children at the same ages.

6. Transitional cells average approximately 2 to 3 per cent., not varying greatly at the different ages.

7. Mastzellen, about 0.3 to 0.6 per cent. Frequently absent.

TABLE 2.—LEUKOCYTE PERCENTAGES IN CHILDREN (KARNITSKY)

Age, Years	Total Leukocyte Count	Percentage of Neutrophils	Percentage of Lymphocytes	Percentage of Eosinophils
1 to 2.....	12,500			
2 to 3.....	9,400	40.7 to 30.2	60 to 75.6	
3 to 4.....		40.3 to 32.1	58 to 45	
4 to 5.....		39.7 to 34	53 to 45	Varies
5 to 6.....		59 to 35	52 to 32	Greatly.
6 to 8.....	7,900	51 to 37.3	52 to 35	3.8 to 8.6
8 to 10.....		62.6 to 40	46.6 to 24	
10 to 12.....		66.6 to 43.7	39 to 22	
12 to 14.....		63.5 to 47.6	40 to 23	

TABLE 3.—LEUKOCYTE PERCENTAGES IN CHILDREN (CARSTANJEN)

Age.	Neutrophils, Per Cent.	Lymphocytes, Per Cent.	Eosinophils, Per Cent.	Transitionals, Per Cent.	Large Mononuclears, Per Cent.
1 to 6 mos.	34.5	50	3.5	10.5	0.5
6 to 12 mos.	40.8	49.2	0.7	8.2	0.9
1 to 2 yrs..	41.9	47	3	7.5	0.4
2 to 3 yrs..	48.2	38.4	3.9	8.7	0.7
3 to 4 yrs..	52.6	33.2	5.7	7.9	0.5
4 to 5 yrs..	61	25.8	6.3	7.2	0.3

8. Large mononuclear neutrophils, 1 to 3.3 per cent. About the same at the different ages.

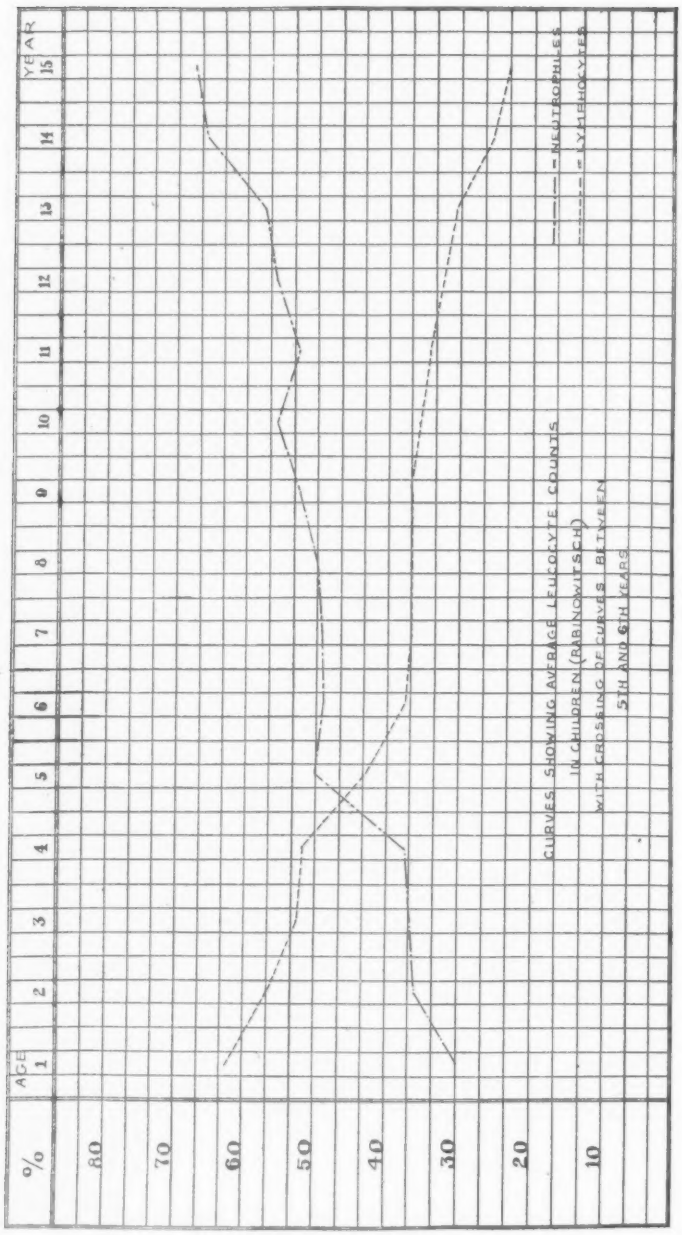
The predominance of lymphocytes in the hematology of the infant is an exceedingly interesting and important question. The probability is that the lymphocytes do predominate in the normal infantile blood, but it should be demonstrated that these children are perfectly normal at the time of examination. It is certain that in the acute infections the lymphocytes are early replaced by the polymorphonuclear lymphocytes.

It has been with considerable reluctance that I have convinced myself of the continued high lymphocyte count as described in the various monographs on the blood in late infancy and early childhood, in the face of the work here presented. Certain it is that early in normal childhood the curves simulate the adult counts; but nevertheless it is quite true that many illnesses more common to early than to adult life do involve the lymphatic system, and thereby stimulate these organs to cell functions which dominate the blood-picture. I desire to present seemingly convincing evidence of the similarity of the reaction to acute infection in infancy, childhood and adult life.

Leukocytosis in children is of extremely common occurrence, often arising from causes of the most trivial character and developing to a greater degree and much more rapidly than in the adult. It is, therefore, to be regarded with less significance than when it occurs in the mature. Usually the polymorphonuclear cells are chiefly involved in the increase, but the inclination of the blood of children to revert to the embryonic type appears to cause in many cases a disproportionate increase of lymphocytes in relation to the other forms, this peculiarity being especially true of the various pathological leukocytoses not due to the acute infections. Physiological leukocytosis in children usually affects chiefly the polymorphonuclear neutrophil cells.

BLOOD EXAMINATIONS IN EPIDEMIC CEREBROSPINAL MENINGITIS

Rusca, in his article, gives the results he obtained from repeated blood examinations in cerebrospinal meningitis and quotes from them in nine cases; six of which were fatal and three in which there was recovery, and all of which were treated symptomatically without the use of serum. This group of cases is of especial interest when comparisons are made with my group of cases—all of which were treated with Flexner serum—both as to the effect of the serum and of anaphylaxis on the blood-picture in the cases which ended in recovery and in the fatal case. In this article Rusca refers to work on the blood in epidemic cerebrospinal meningitis by Türk, Schindler, Zand and others. They all found a more or less marked leukocytosis with a predominance of the polymorphonuclear leukocytes, but they are not agreed as to the prognostic importance of the disappearance of the eosinophils, some believing that the disappearance was of ill omen, others believing it to be a good sign. The therapy practiced in Rusca's cases consisted of repeated lumbar punctures, large doses of sodium salicylate, ice packs, caffein and nutrient enemas. He finds in all cases a more or less marked leukocytosis. The polymorphonuclear leukocytes show the greatest increase. The lymphocytes are increased in some cases, decreased in others. With each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CURVES SHOWING AVERAGE LEUCOCYTE COUNTS
IN CHILDREN (RABINOWITSCH)
WITH CROSSING OF CURVES BETWEEN
5TH AND 6TH YEARS
= NEUTROPHILES
= LYMPHOCYTES

Chart I.

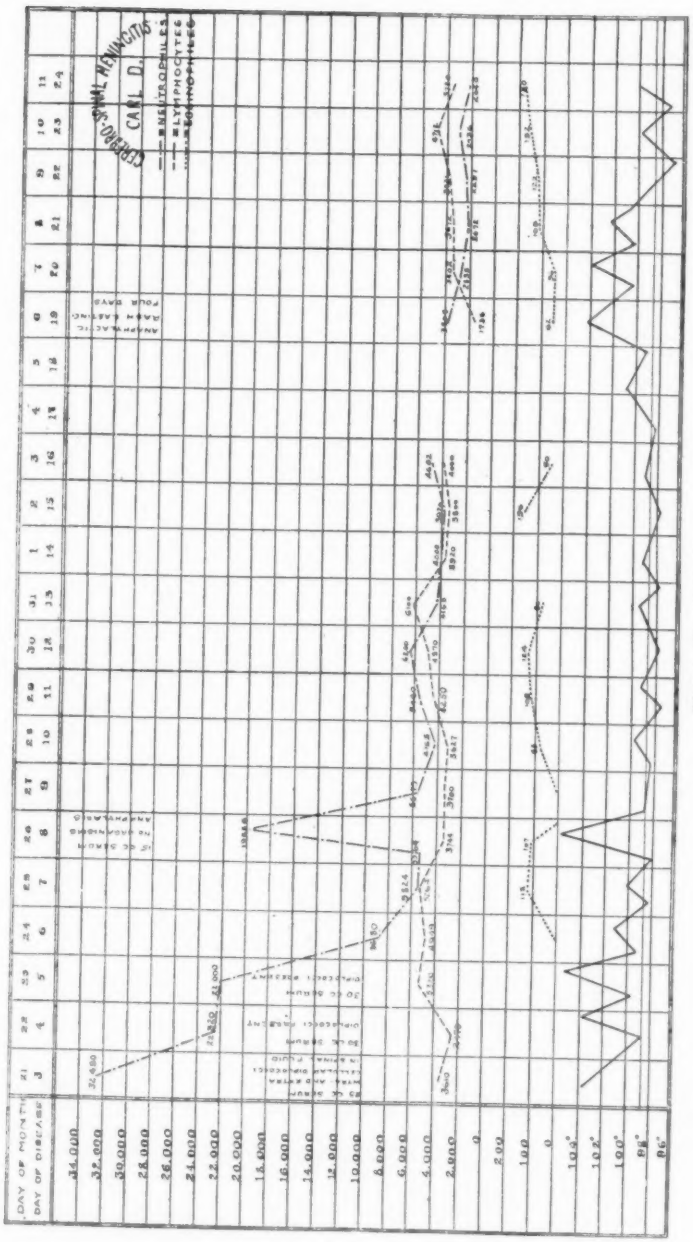

Chart II—Case 1.

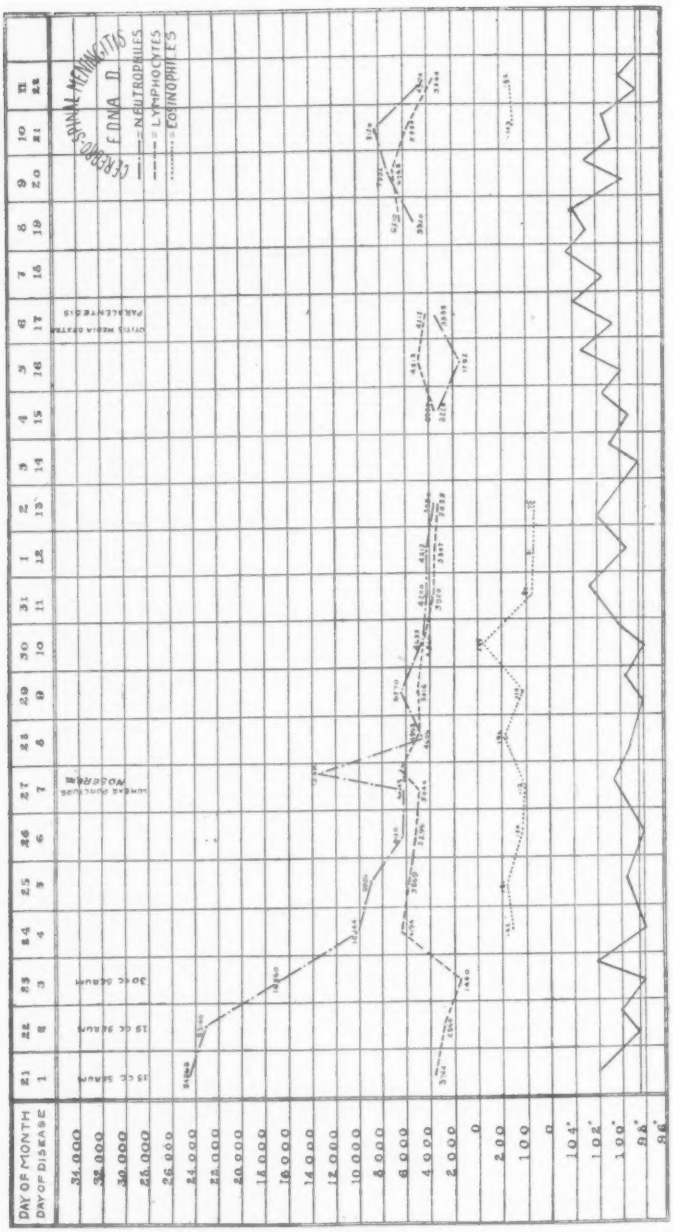

Chart III—Case 2.

exacerbation there is an increase in the percentage as well as the absolute number of neutrophilic leukocytes and the reverse is true in improvement. With the lymphocytes the opposite prevails. This results in a separation of the two curves during the recrudescence and an approximation and often a crossing during convalescence. In Rusca's three cases which ended in recovery, the curves crossed. In the first case, this occurred on the thirty-sixth day of the illness. In the second, it followed very rapidly on the first lumbar puncture, which was made during the fourth week of illness. In the latter case, before lumbar puncture, the polymorphonuclear leukocytes were 12,741, the lymphocytes 2,132, with

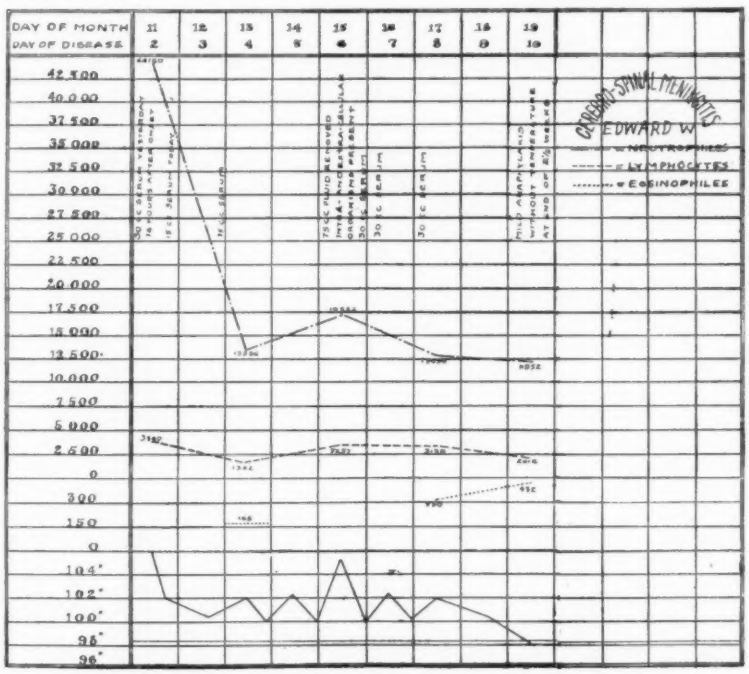

Chart IV—Case 3.

no eosinophils or basophils. Two days later there were 3,227 polymorphonuclears, 3,500 lymphocytes, 35 eosinophils and 28 mast cells. He states that had serum been used in this case, it would have been erroneously credited with this marked change. He also notes a measles-like rash in the first days of the illness in this case, whose occurrence had been previously described by Curtis and who states that it is of good import. Block, Curtis and others suggest that the presence of a hemorrhagic rash, in contradistinction to the measles-like rash, is of bad import. In Rusca's third case, crossing of the curves occurred on the ninth day. In all of these cases the crossing of the curves was due to two factors:

an absolute decrease in the polymorphonuclears and an absolute increase in the lymphocytes. The eosinophils were markedly increased in two of his cases during convalescence, in each to 6.5 per cent. In the third case they never were more than 216 per cubic centimeter, or 2.5 per cent. In the fatal cases the eosinophils were either very much decreased or absent during the acute stage and were always absent before death. The mast-cells vary, being absent in some of the cases during convalescence, increased during the course of one of his fatal cases, but always absent before death.

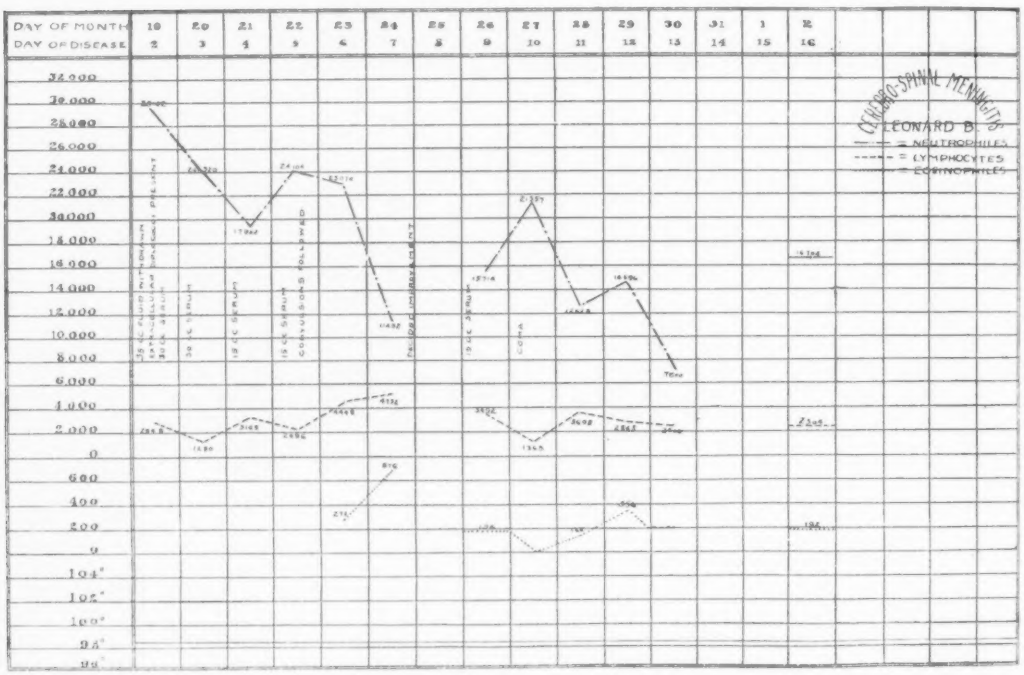

Chart V—Case 4.

He concluded that at the beginning there is always a leukocytosis associated with an absolute increase in the polymorphonuclear leukocytes. During the course of the illness a separation of the polymorphonuclear and leukocyte curves occurs and during convalescence an approximation. In all of his cases that ended in recovery, the curves crossed, and were associated with a normal or increased eosinophil count. In the lethal cases there was no crossing of the curves and the eosinophils and mast-zellen always disappeared before death.

Of my series of six cases, five patients recovered and one died.

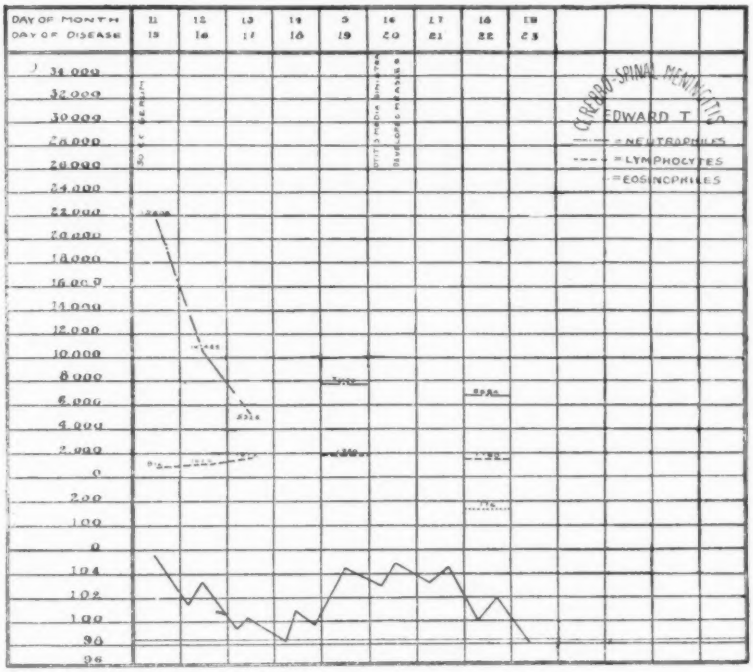

Chart VI—Case 5.

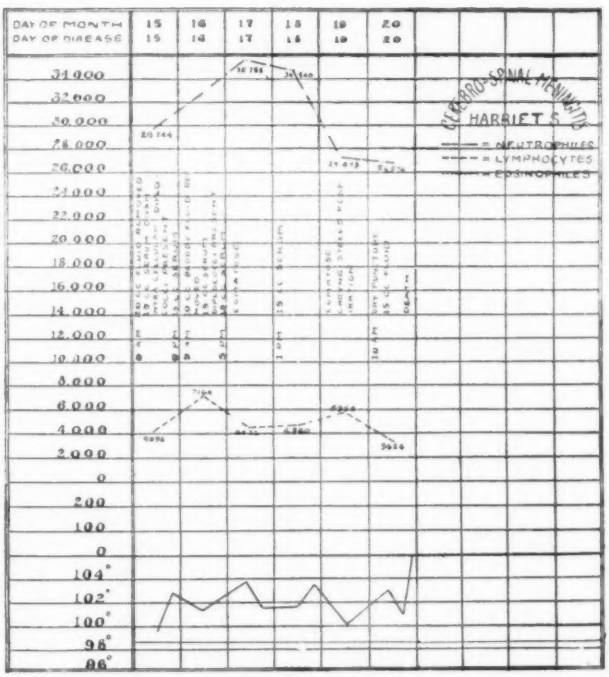

Chart VII—Case 6.

SUMMARY OF FINDINGS IN PRESENT SERIES OF CASES OF EPIDEMIC MENINGITIS

1. Total Leukocyte Counts

In Case 1, the patient was seen on the third day, suffering from a very virulent form of meningitis associated with delirium, extreme rigidity, etc. The total count before treatment was 36,100.

In Case 2, the patient, aged 5, a sister of the patient in Case 1, who was seen during the first eighteen hours of the illness, was still rational, and had a total count of 28,800.

In Case 3, the patient, aged 7, was seen within twelve hours of the onset, and had a total white count of 48,000. The child was at this time wildly delirious. In this case the count was not taken until twelve hours after the first 30 c.c. of serum were given.

Case 4, in which the symptoms were very violent, was seen twenty hours after the first vomiting, and four hours after the first slight convulsion. This patient remained in coma for fully three weeks. The total white count was 31,650 within twenty hours of the first attack of vomiting.

Case 5, in which the patient was aged 2 years and 9 months, was apparently a subacute case with a history of vomiting two weeks previously and a gradually increasing opisthotonos, rigidity, etc., and entered the hospital with white count of 22,800.

Case 6, in which the patient was aged 2, was very severe, the patient entering the hospital with a history of convulsions two weeks previously, with which were associated opisthotonos, rigidity, diarrheas, etc. The white count was 33,800.

As will be seen, all of these cases presented a high leukocyte count. In the first four, all of which recovered and which were seen very early in the attack, the number passed 30,000 and went as high as 48,000. In the fifth case, a subacute one, resulting in recovery, the count was 22,800, and the last case, also subacute, but with fatal issue and a complication of diarrhea, showed 33,800. Therefore, we may conclude that all of our series were associated with a marked leukocytosis.

2. Percentage of Neutrophils

Of this high leukocyte count, the polymorphonuclear neutrophils average in the first case, 90 per cent.; in the second, 80 per cent.; in the fourth, 92 per cent.; in the fifth (subacute case), 82 per cent., and in the sixth (subacute case with death), 84 per cent. This conclusively shows that the polymorphonuclear neutrophils were the predominating cells in these first counts made before serum was given.

3. Course of Neutrophils

In Case 1 the polymorphonuclear cells, numbering 32,490 before the first serum, dropped to 22,300 within twenty-four hours after the first 25 c.c. were given and to 8,680 after 85 c.c. and within three days after the first treatment. In this case, the injection was guided by the finding of both intra- and extracellular organisms up to the time of the injection of the third dose of serum.

Case 2. The 24,568 neutrophils present on the first examination dropped to 23,000 after the first 15 c.c. of serum, to 16,500 after the second 15 c.c. and to 10,500 after the injection of a total of 60 c.c.

Case 4 showed a polymorphonuclear count of 29,000 before the first 30 c.c. and 24,300 following the first. On the fifth day the count again rose to 24,000 and was associated with marked convulsions. The seventh day there was a decided improvement, with a fall to 11,492, two days after the patient had had his last serum, a total of 90 c.c. Another 15 c.c. was given on the ninth day, which was followed by more marked spasm and more intense coma, with a rise of the neutrophils to 15,714. Following this, there was gradual improvement and they again dropped to 7,600.

Case 3, with 44,160 polymorphonuclear leukocytes before the serum was given, dropped to 13,386 within forty-eight hours after the injection of 60 c.c. However, on the sixth day there was again a rise to 19,822, and on examination of the spinal fluid, it was found to contain both intra- and extracellular organisms. Sixty c.c. were again injected, 30 c.c. on each of the two subsequent days and the neutrophil count fell to 11,952. This was followed by complete recovery. On the sixth day with the return of the organisms, there was a rise in temperature to 105.5, followed by lesser rises on the two succeeding days to 102, but associated with steadily decreasing polymorphonuclear and lymphocyte counts and the reappearance of eosinophils following their disappearance with a recurrence of the organisms on the sixth day.

Case 5, complicated by the occurrence of measles, entered the hospital with a count of 19,608 neutrophils. Thirty c.c. of serum were given and within forty-eight hours the count dropped to 5,322. On the fifth day in the hospital the neutrophils reached 7,920, and on the sixth day the child developed measles. The count made on the eighth day and when the measles eruption was at its height, showed 6,864 neutrophils, and for the first time a return of the eosinophils. This case is of importance because of the influence which the measles may have had on the count. Rusca and others have described a measles-like eruption in some favorable cases, but they do not state specifically whether or not these cases were true measles. In passing, it is of interest to note that in this severe subacute case, in which the child was undoubtedly exposed to measles after the onset of the meningitis, the influence of the measles

on the course of the latter was favorable, if it influenced it at all. It might also be noted that with the onset of the prodromata and subsequently the rash of the measles, the temperature rose to 104.5, and reached this point on three subsequent days. As in the cases with an anaphylactic reaction, in which the temperature rose to a considerable degree, there was no reaction on the part of the white count.

Case 6 showed 29,744 neutrophils on entering, steadily rising to 35,778 on the third day in the hospital, notwithstanding the fact that 60 c.c. of serum had been given. At this point the child was in deep coma. Following this, another 30 c.c. of serum were administered, and the neutrophil count dropped to 26,576. Death ensued without the overcoming of the high neutrophil count. This case might be likened to some of the pneumonia cases which show an extremely high count in the neutrophils in the presence of very virulent infection and fatal result.

4. Lymphocyte Count

Case 1 had the highest total lymphocyte count, 6,100 on the tenth day following the serum, as compared with 3,610 on the day the serum was given.

In Case 2, the highest lymphocyte count was 6,400 on the seventh day after the serum, as compared with 3,744 before the serum was given. In this case they rose as high as 6,768 on the twenty-first day. In these two cases with the disappearance of the organisms, the total lymphocytes became about double the number seen before serum was administered. While the percentage of large lymphocytes in Case 1 was reduced to 1 per cent. before reaction to the serum, in the lighter case, Case 2, they did not decrease below 4 per cent. In Cases 3 and 4, although there was an absolute increase in the lymphocytes, slightly more marked in Case 4 than in Case 3, in neither of these cases was there any great increase, the highest point reached in Case 4, being 4,732, and in Case 3, 3,840.

Case 5, the one complicated with measles, showed a decrease from the normal rather than an increase at any time.

Case 6. In this, as with the neutrophils, there also was a constant increase in the absolute lymphocyte count, not due, however, to any marked increase in the percentage of the cells as seen in the blood, but to the marked general increase in the white count. Therefore, in all of these cases, except the one complicated by measles, we see a true lymphocytosis, however, in no case a marked one.

5. Relation of the Neutrophils to the Lymphocytes During Convalescence

In Case 1 we see a very close approximation of the two curves on the fifth day following serum and the crossing of the curves on the tenth day.

In Case 2 we again see a very close approximation on the fifth day following the serum and the crossing of the curves on the eighth day.

In Cases 3, 4 and 5, while the curves are not carried along for a sufficient time to allow of a very close approximation of the curves, there was a tendency on their part to converge as soon as the result of the serum was manifest. This was not so in Case 6, in which there was a tendency to diverge up to the time of death.

6. Eosinophils

Only once in all of my counts did the eosinophils reach 4 per cent. of the total cells counted, and only occasionally as high as 3 per cent. In all cases during the acute state and before the serum reaction, there was a complete disappearance of eosinophils. In Case 1, they did not reappear until 90 c.c. of serum had been given, and they disappeared again for two days following the reaction on the fifth day. Following this they remained constant, the count ranging from 50 to 180 to the cubic millimeter, but at no time during the twenty-one days of this case did they reach what might be considered a normal count.

In Case 2, they also remained absent until the fourth day and until 60 c.c. of serum had been given. Following this, they remained constant, ranging from 1 per cent. to 3 per cent. during the following nine days, when they again disappeared for several days during the development of an otitis media. The highest count was 3 per cent, or 285 cells, on the tenth day following the first serum. More commonly they ranged between 80 and 196, never passing what might be considered the normal range in the healthy child.

In Case 1, although they did not entirely disappear during the period of anaphylaxis, the lowest count during the whole convalescence was found at this time.

In Case 4 they remained absent until 90 c.c. of serum had been given, on the fifth day they reached 278, and, with a decided improvement on the sixth day, they numbered 676. As in Case 1, with the injection of serum on the eighth day there was a marked increase in the leukocytes with a corresponding decrease in the lymphocytes and a disappearance of the eosinophils. They again reappeared during the period of improvment and remained quite constant throughout the convalescence.

In Case 3 they appeared on the third day following the serum, again to disappear with the recurrence of the meningococci in the spinal fluid, remaining away until the eighth day, when they reappeared, ranging in number from 330 to 532 and staying constant during the balance of the time.

In Case 5, the one associated with measles, the eosinophils remained absent until the eighth day after the serum, when they appeared, 176 to the cubic millimeter.

In Case 6, with a fatal issue, there never was any reappearance of the eosinophils during the stay in the hospital.

CONCLUSIONS AS TO EPIDEMIC MENINGITIS

1. All of my cases were associated with a marked leukocytosis.

2. The neutrophils were always the predominating cell before the administration of serum.

3. There was a moderate but absolute lymphocytosis in all cases, most marked during convalescence, except in the one infected with measles. It was most marked in the fatal case.

4. There was always an approximation and in some a crossing of the neutrophil and lymphocyte curves during convalescence.

5. Eosinophils disappear absolutely very early in the infection and do not recur until convalescence has set in. With recrudescence and the reappearance of organisms in the spinal fluid, they again disappear. Following the withdrawal of spinal fluid and the injection of serum, there may be an increase in the total white and neutrophil count with a decrease of eosinophils, but in our experience, although the late anaphylactic reactions may be associated with a rise in temperature, the polymorphonuclear cells remained low and eosinophils present. Although there may be a distinct increase occasionally, at no time in our series of cases was there a marked eosinophilia. Their presence and reappearance in our cases was always associated with signs of good omen.

6. Every case must be studied individually with reference to the findings presented, not only as to the presence of meningitis, but the possibility of secondary infections.

7. Cellular blood examinations have no diagnostic value. Lumbar puncture is the source of accurate information.

8. Much can be learned as to prognosis by regular examination of the leukocytes. A drop in the neutrophil count with an increase in the lymphocytes and a reappearance of the eosinophils indicates a good prognosis. An approximation of the neutrophil and lymphocyte curves is of good import.

9. The leukocyte count is of most importance as an indicator for further prognostic punctures or the need of serum. An increase of the neutrophils and temperature with disappearance of the eosinophils should always lead to further spinal puncture.

10. The temperature and cerebral reactions often associated with late anaphylaxis can be interpreted by careful leukocyte counts, and the further injection of serum, with its accompanying dangers, may be avoided. The neutrophils remain low and the eosinophils usually are present during this stage.

11. Serum should always be injected by the gravity method.

A STUDY OF THE BLOOD-PICTURE IN PNEUMONIA

Believing that a careful study of the general blood-picture, as well as the differential cell counts, would be of considerable value in the

differential diagnosis of pneumonia, in forecasting the prognosis as to recovery and the development of complications, and also in establishing the diagnosis of the type of pneumonia in young infants, that is, the differentiation between lobar and bronchopneumonias, a series of fifty cases of pneumonia of different types and associated with various complications were selected for this work. The cases have been arranged in a series of tables, according to type and complications, and each one is accompanied by an individual summary of the salient features of that group. Several interesting conclusions of practical value in the classification, differential diagnosis, prognosis and complications of the pneumonias have been reached by these analyses.

LOBAR PNEUMONIA

Series 1.—Uncomplicated Pneumonia Under 1 Year of Age

SUMMARY OF TABLE 4

The two cases in Table 4 were the only ones of uncomplicated pneumonia in my series, and both were of the lobar type, which is worthy of notation, because of the frequent classification of pneumonia under 2 years as bronchopneumonia. In Case 1, the neutrophils never fell below 62 per cent., nor the combined lymphocytes above 35 per cent. As in the older infants and children, the eosinophils returned with convalescence. In Case 1, there was tendency on the part of the lymphocytes to remain high after beginning convalescence. In the second case the neutrophils were 82 per cent. at the height of the infection.

These findings are of interest because of the marked reaction of the polymorphonuclear leukocytes to the pneumococcus infection, just as one would expect in older healthy children, notwithstanding the normal lymphocytosis of infant blood. These findings differ from Cases 32, 34 and 35, aged, respectively, 5½, 6 and 2 months, which were secondary pneumonias in rachitic and anemic infants. In all three of these cases there was a distinct lymphocytosis. In Case 37, a ten-week-old infant with a double lobar pneumonia and a secondary empyema, there was again the distinct reaction seen in the infections of older individuals, namely, 53,800 leukocytes, with 76 per cent. of neutrophils, 24 per cent. of combined lymphocytes and no eosinophils.

This series of six cases leads me to the following conclusions:

1. Primary pneumonias of early infancy are commonly of the lobar type.

2. Young infants react to infections with a high neutrophil count, the same as is seen in older children.

3. Eosinophils remain absent in young infants during the height of infections.

4. Bronchopneumonias are usually either secondary pneumonias or occur in infants with low vitality.

SUMMARY OF SERIES 2, TABLE 5

1. The primary pneumonias of Series 2 were of the lobar type.

2. In well-nourished infants between the first and second years, the larger the amount of lung tissue involved, the greater the tendency to a high leukocytosis.

3. Anemic and poorly-nourished infants usually show lower total leukocyte counts.

4. The drop in the total white counts as well as the neutrophil count following convalescence is usually less rapid than in older children. This is interesting when we consider the normal high lymphocyte count of infants.

5. In uncomplicated cases the eosinophils usually returned on the day of the crisis or in one to four days following, but rarely in numbers exceeding the normal limits. In the complicated cases, they were slow in reappearing.

CONCLUSIONS AS TO SERIES 3 AND 4, TABLE 6

1. All were lobar pneumonias.

2. A true leukocytosis was present in all cases, highest in the uncomplicated cases.

3. Neutrophils were always the predominating cells before the crisis, and in all uncomplicated cases fell rapidly after the crisis.

4. In all cases there was a true lymphocytosis comprising both large and small cells, the small cells, however, predominating. Following the crisis they rapidly dropped to near the normal in most instances.

5. In the nineteen cases of these two series the eosinophils returned in Case 13 (with two previous attacks of pneumonia) four days before the crisis; in one, three days before; in two, two days before; in one, one day before; in three, on the day of the crisis; in two, two days after, and in one, three days after the crisis. In three complicated cases none reappeared in the counts made.

In only one instance, Case 21, did the lymphocyte curve cross the neutrophil curve.

SUMMARY OF SERIES 5, TABLE 7

1. Bronchopneumonia in Series 5 was either secondary in poorly-nourished infants or followed by complications. Three died and one recovered.

2. The total leukocyte counts varied with the associated findings and complications.

3. There was a constant tendency to high leukocyte counts.

4. Eosinophilia was present in two cases and absent in the other two cases.

SUMMARY OF TABLE 8

1. High total white count usually following an earlier decrease after crisis or lysis.

2. High neutrophil count and absence of eosinophils.

SUMMARY OF TABLE 9

1. None of the cases with serous effusions showed much over 20,000 total counts, and one was as low as 11,600. The neutrophils were constantly high.

2. The large lymphocytes varied, but there was no tendency toward a lymphocytosis, although all gave positive tuberculin tests, and the eosinophils were absent in all cases at the time of examination.

SUMMARY OF TABLE 10

1. High total leukocyte count.

2. Low neutrophil and high small lymphocyte count.

3. Absence of eosinophils.

SUMMARY OF TABLE 11

1. If pus infection precedes the crisis, the total white and neutrophil counts remain high.

2. If infection becomes manifest following the crisis and the count has dropped, there reappears an absolute neutrophil increase.

3. With secondary infection, eosinophils usually disappear, if present, following the crisis.

SUMMARY OF TABLE 12

1. Eosinophils were slow in disappearing during the early part of the illness and returned the day following the crisis in normal numbers.

A case to compare with the one in my series showing worms is Professor Lichtenstern's case of ankylostomiasis. In 1897, the percentage of eosinophils in the blood was 72. The patient acquired croupous pneumonia during the acme of which the number of eosinophils dropped to 6 or 7 per cent., then rose to 54 per cent. during convalescence from the pneumonia. After the removal of the worms the number fell at once to 11 per cent. In 1898, when the ankylostomata were few and Charcot crystals were no longer present in the feces, the number of eosinophils amounted to 7 per cent.

Arneth, in a series of cases examined for eosinophilia during the course of pneumonia, arrives at the following conclusions:

Eosinophils were present in 50 per cent. of eleven cases examined during the course of the pneumonia. Fifty per cent. of these showed eosinophils, in very limited numbers, however. They were cases of very short courses and abortive cases and in one a chronic case (the only one

of his series). He concludes that the severer the case the more certain are we of a complete disappearance of eosinophils. Shortly after the chill we may find an occasional one. They usually reappear (seven cases) on the day following the crisis, in one case before the crisis. In nine cases there was a post pneumonic eosinophilia; in six cases there was no increase above normal, in most of the former the normal per cent. was only occasionally passed.

Arneth believes that eosinophils play no rôle in the fight against the bacterial invasion or the development of the antibodies in pneumonia. He also believes that Türk must change his view that the eosinophils, as do the leukocytes, play an important rôle in the fight against the invasion. Arneth bases his belief on the continued absence of eosinophilia in some cases or its late appearance long after the crisis in others. The presence of eosinophilia is very varied in different cases. From his one chronic case in which the eosinophils persisted throughout the course, he raises the question as to whether they do not in time become accustomed to the infection, this explaining their constant presence (one case seems too little to judge), but my Case 41, in which there had been two previous attacks, gives practically the same findings. A need of them results in their recurrence in greater or less numbers. Arneth does not believe that their return is due to chemotactic influences. The above series of Arneth were aged 42, 28, 58, 64, 44, (?), 37, 54, 33, 18 and 10 years, respectively.

Naegeli states that eosinophils are absent or rarely present until near the time of the crisis. Often the first ones are found one or two days before the crisis. They increase more rapidly after the temperature drops until we actually have a post infectious eosinophilia. In itself this is a sign of good omen as it indicates that the intoxication is decreasing.

Japha states that in the postfebrile period of pneumonia there is frequently found an increase of eosinophils, and sometimes an outspoken eosinophilia, though as a rule the increase is moderate. From a clinical standpoint, all that has been learned is that the ordinary metabolic products of bacteria repel the eosinophil cells.

Türk believes that the eosinophils in bacterial infections play a rôle directly opposite that of the neutrophils. They disappear entirely from the blood-picture or are present in very small numbers. In croupous pneumonia they usually return again shortly before the crisis. During convalescence a distinct and absolute increase can often be demonstrated, although this rarely reaches a high degree. An early and marked increase in pneumonia frequently indicates an abortive type. Türk further states that large mononuclear leukocytes and transitionals run

TABLE 4.—Series 1. Uncomplicated Pneumonia Under 1 Year of Age

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count												Date of Crisis	Laboratory Examination	Prognosis
								Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent			
1	5/12	Lottie H..	Right upper.....	4	2/26/13	103	14,000	8,680	62	4,200	30	700	5	420	3	...	....	...	....	.....	Urine: alb +	
					2/27/13	103	25,000	15,500	62	7,750	31	1,000	4	750	3	...	....	...	....	.....		
					3/ 1/13	101	21,000	15,120	70	5,184	24	864	4	432	2	...	....	...	....	Lysis.		
					3/ 2/13	100	19,900	13,532	68	5,771	29	398	2	.....	..	199	1	...	....	3/ 1/13		
					3/ 3/13	100	26,600	16,758	63	9,576	36	.....	..	266	1	...	....	...	....	.....	.....	Improved.
2	8/12	Sam H....	Involvement of right and left upper lobes.	5	7/13/13	106	37,600	30,832	82	6,016	16	752	2	.....	..	...	...	...	....	7/17/13	.....	Cured.
					7/16/13	104.8	35,000	30,200	84	4,060	14	740	2	.....	..	...	....	...	....			
					7/18/13	99	12,000	7,800	65	3,000	25	840	7	.....	..	360	3	...	....			

TABLE 5.—Series 2. Pneumonias in Children 1 to 2 Years of Age

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count												Date of Crisis	Laboratory Examination	Prognosis
								Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.			
3	1¼	Issy R...	Left upper and lower and right lower. Complications: acute cardiac dilatation.	4	1/26/13	104.5	28,000	14,560	52	1,240	43	1,120	4	280	1	...	....	...	....	.....	Urine: albumin, trace.	
					1/27/13	105.5	32,000	20,800	65	9,600	30	1,600	5	.....	..	...	....	...	....			
					1/28/13	106.8	20,000	14,200	71	4,600	23	800	4	400	2	...	....	...	....			
					1/29/13	105.8	21,000	14,700	70	4,200	20	1,260	6	420	2	...	....	420	2			
					1/30/13	106.2	19,000	12,920	68	4,180	22	1,330	7	570	3	...	....	...	....			
					1/31/13	105	24,000	15,120	63	5,520	23	1,960	9	960	4	...	....	...	....			
					2/ 1/13	105	29,200	20,440	70	5,548	19	2,920	10	292	1	...	....	...	....			
					2/ 2/13	103.6	20,300	13,804	68	4,060	20	2,030	10	406	2	...	....	...	....			
					2/ 3/13	102	28,000	22,680	81	4,200	15	1,120	4	.....	..	...	....	...	....			
					2/ 4/13	103	35,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....	.....	.....	Died.
4	2/12	Rosie L...	Left upper and lower lobes involved.	2	3/28/13	105	22,000	19,580	89	1,540	7	660	3	220	1	...	....	...	....	.....	Urine: alb. +	
					3/29/13	104	25,200	21,672	86	2,268	9	756	3	504	2	...	....	...	....			
					3/30/13	103	22,800	20,520	90	1,368	6	456	2	228	1	...	....	228	1			
					3/31/13	103	25,000	21,750	87	2,000	8	1,000	4	250	1	...	....	...	....			
					4/ 1/13	102	22,200	17,538	79	4,440	20	222	1	.....	..	...	....	...	....	4/ 1/13		
					4/ 2/13	99	26,400	20,592	78	4,224	16	1,056	4	264	1	...	....	264	1	4/ 2/13		
					4/ 3/13	100.4	24,200	19,602	81	3,388	14	968	4	.....	..	...	....	242	1			
					4/ 4/13	99	20,000	15,200	76	3,600	18	800	4	200	1	...	....	200	1			
					4/ 5/13	100	15,000	10,800	72	3,000	20	900	6	.....	..	...	....	300	2			
					4/ 6/13	98.6	12,000	8,760	73	2,280	19	600	5	240	2	...	....	120	1			
					4/ 7/13	99	14,500	11,165	77	2,900	20	290	2	.....	..	...	....	145	1			
					4/ 8/13	.....	12,400	8,804	71	2,850	23	620	5	.....	..	124	1	...	....			
					4/ 9/13	.....	13,100	9,039	69	2,882	22	655	5	262	2	131	1	131	1	.....	.....	Cured.
5	2	Bella S...	Left lower lobe. Complication: delirium+++	7	1/13/13	103.8	33,600	.....	..	.....	..	.....	..	.....	..	...	....	...	....	.....	Urine: alb. trace.	
					1/14/13	105	47,000	37,130	79	8,460	18	940	2	470	1	...	....	...	....			
					1/15/13	106	44,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					1/16/13	98.4	24,000	19,200	80	3,360	14	720	3	.....	..	720	3	...	....	1/16/13		
					1/17/13	98.4	22,000	17,600	80	3,300	15	880	4	.....	..	220	1	...	....			
					1/18/13	99	20,000	14,800	74	3,400	25	1,200	6	400	2	200	1	...	....	.....	.....	Cured.
6	1	Lois P....	Right upper, middle and lower lobes; also left upper and lower. Complication: otitis media.	4	12/26/13	104.5	30,000	21,000	70	7,500	25	1,200	4	300	1	...	....	...	....	.....	Urine: alb. + Von Pirquet neg.	
					12/30/13	105.5	48,500	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					1/ 2/13	105	63,000	42,210	67	17,010	27	3,150	5	630	1	...	....	...	....			
					1/ 4/13	105	38,500	24,255	63	12,320	32	1,155	3	770	2	...	....	...	....			
					1/ 5/13	104	48,300	30,429	63	15,456	32	1,932	4	483	1	...	....	...	....			
					1/ 7/13	104.8	46,600	28,426	61	15,844	34	1,864	4	466	1	...	....	...	....			
					1/ 8/13	101	36,300	23,595	65	10,890	30	1,089	3	363	1	363	1	...	....			
					1/ 9/13	101.5	33,600	17,472	52	14,784	44	1,008	3	336	1	...	....	...	....			
					1/10/13	101	31,000	18,600	60	11,780	38	620	2	.....	..	...	....	...	....			
					1/11/13	102	32,400	21,384	66	9,720	30	972	3	324	1	...	....	...	....			
					1/12/13	102	30,600	18,584	64	9,792	32	918	3	306	1	...	....	...	....			
					1/13/13	101	30,400	20,064	66	8,816	29	1,216	4	304	1	...	....	...	....			
					1/14/13	101	28,900	18,207	63	9,537	33	867	3	289	1	...	....	...	....			
					1/15/13	.....	26,300	15,780	60	8,205	35	789	3	263	1	263	1	...	....			
					1/17/13	.....	18,000	10,800	60	5,400	30	200	5	540	3	360	2	...	....	.....	.....	Cured.
7	2	Morris S..	Left upper and lower.	2	3/23/13	.....	37,200	31,248	84	4,092	11	744	2	1,116	3	...	...	...	....	3/24, 25	Urine: alb.	Cured.

					1/15/13	.....	26,300	15,780	60	8,205	35	789	3	263	1	263	1	...	....				
					1/17/13	.....	18,000	10,800	60	5,400	30	200	5	540	3	360	2	...	....	.....	.....		
7	2	Morris S..	Left upper and lower. Complication: otitis media.	2	3/23/13	.....	37,200	31,248	84	4,092	11	744	2	1,116	3	...	...	...	....	3/24, 25	Urine: alb. trace.	Cured.	
					3/25/13	.....	.....	.....	73	.....	20	.....	6	.....	1	...	....	...	....				
					3/27/13	.....	.....	.....	69	.....	25	.....	2	.....	3	...	1	...	....	.....			
8	2	Bennie R.	Right upper, middle and lower lobes.	4	3/27/13	103.5	33,400	25,718	77	4,676	14	668	2	2,338	7	...	...	...	....	.....	.....		
					3/29/13	104	36,400	28,028	77	6,188	17	1,092	3	364	1	728	2	...	....	3/28, 29	.....	Cured.	
9	2	Ida C.....	Left lower.....	3	12/29	101	20,400	14,280	70	4,896	25	1,020	5	204	7	...	....	...	....	Lysis 1/11	.....	Cured.	
10	1	Gertrude W.	Left upper. Complications: delirious, gonorrheal vag.; poor resistance.	3	12/30/11	†	25,600	13,568	53	9,728	38	1,792	7	.....	..	...	....	512	2	Lysis.	Urine: alb. +.	Cured.	
11	1½	Lilly Z...	Left lower. Complications: partial int. obs. due to adhesions at junction of transverse and descending colon. Pneumococci peritonitis.		6/23/11	104	13,900	10,703	77	2,502	18	417	3	.....	..	139	1	139	1	.....	.....	Died.	

† 101 to 104.5.

TABLE 6.—Series 3, 2 to 4 Years

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count												Date of Crisis	Laboratory Examination	Prognosis
								Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.			
12	3	Solomon K.	Right lower.....	4	1/ 7/13	104	32,000	23,040	72	7,680	24	640	2	320	1	320	1	....	....	.....	Urine: alb. trace.	Cured.
					1/10/13	98	10,500	5,250	50	4,410	42	420	4	105	1	315	3	...	....	1/ 8/13		
13	3	Esther M.	Right apex and left base; two previous attacks of pneumonia.	4	2/27/13	105	13,600	7,616	56	3,954	29	952	7	544	4	544	4	...	....			
					2/28/13	105	14,800	.....	..	.....	..	.....	..	.....	..	...	....	...	....	.....	Urine: alb. trace.	
					3/ 4/13	103	16,600	10,624	64	4,150	25	830	5	996	6	...	....	...	....	3/ 1/13		
					3/ 5/13	101	18,800	12,408	66	4,512	24	940	5	188	1	376	2	376	2			
					3/ 6/13	100	16,900	10,408	62	4,563	27	676	4	507	3	338	2	338	2	.....	.....	Cured.
14	4	Jake S...	Left lower lobe involved..	4	12/ 5/13	105	22,800	20,520	90	1,368	6	912	4	.....	..	...	....	...	....			
					12/ 6/13	106	22,400	20,608	92	896	4	1,344	6	.....	..	...	....	...	....			
					12/ 7/13	105	16,000	13,328	83.3	2,352	14.7	320	2	.....	..	...	....	...	....			
					12/ 8/13	104.6	14,000	12,040	86	1,540	11	420	3	.....	..	...	....	...	....			
					12/ 9/13	104.9	13,000	9,776	75.2	2,600	20	442	3.4	182	1.4	...	....	...	....			
					12/10/13	98.6	14,000	9,520	68	4,060	29	420	3	.....	..	...	....	...	....	12/ 9/13		
					12/11/13	98.6	12,600	8,190	65	4,284	34	126	1	.....	..	...	....	...	....			
					12/12/13	99	16,300	10,758	66	5,053	31	489	3	.....	..	...	....	...	....			
15	2½	Jacob A..	Left upper and lower. Complication: rachitis and anemia.	4	5/ 7/13	105	32,600	23,795	73	4,238	13	2,608	8	1,956	6	...	....	...	....	.....	.....	Cured.
					5/ 9/13	106	28,100	19,670	70	3,934	14	1,967	7	1,967	7	281	1	281	1	5/ 9/13		
					5/11/13	99	20,700	14,076	68	3,312	16	1,863	9	1,449	7	...	....	...	....			
					5/12/13	99.5	23,600	16,520	70	4,248	18	1,416	6	1,416	6	...	....	...	....			
					5/13/13	99.4	27,100	18,157	67	5,691	21	2,168	8	1,084	4	...	....	...	....			
					5/14/13	99.6	31,700	20,605	65	8,242	26	2,219	7	634	2	...	....	...	....			
					5/16/13	99.4	22,100	16,796	76	3,978	18	663	3	663	3	...	....	...	....			
16	6	George W.	Complete consolidation of right upper and middle lobes, with marked cerebral symptoms. Lumbar puncture made.	2	11/ 6/13	...	48,700	45,047.5	92.5	340.9	90.7	3,068.1	6.3	243.50	.5	...	....	...	....	.....	.....	Improved.
					11/ 7/13	...	42,300	.....	..	.....	..	.....	..	.....	..	...	....	...	....		Sp. fluid contained few polys.	
					11/11/13	...	61,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....	11/11/13		
					11/14/13	...	.....	.....	61	.....	32	.....	3	.....	..	...	3	...	....	.....	.....	Cured.

Series 4, Age 4 Years and Up

17	7	Aaron G..	Left lower; later right upper.	2	2/21/12	105.5	22,400	18,368	82	2,016	9	1,344	6	672	3	...	....	...	....	.....	Urine: Alb. trace; Hyalin casts.		
					2/22/12	104.5	39,200	31,752	81	3,136	8	2,352	6	1,568	4	...	....	...	....				
					2/23/12	104.5	38,800	32,592	84	3,492	9	2,328	6	388	1	...	....	388	1				
					2/24/12	104.5	25,200	21,420	85	2,772	11	756	3	.....	..	252	1	...	....	2/24/13			
					2/26/12	98.6	8,400	6,300	75	1,680	20	336	4	.....	..	84	1	...	....				
					2/28/12	98.2	8,200	5,576	68	1,968	24	492	6	.....	..	168	2	...	....	.....	.....		
18	9	Pearl G..	Right lower																			Cured.	

17	7	Aaron G..	Left lower; later right upper.	2	2/21/12	105.5	22,400	18,368	82	2,016	9	1,344	6	672	3	...	....	...	....	.....	Urine: Alb. trace; Hyalin casts.	
					2/22/12	104.5	39,200	31,752	81	3,136	8	2,352	6	1,568	4	...	....	...	....			
					2/23/12	104.5	38,800	32,592	84	3,492	9	2,328	6	388	1	...	....	388	1			
					2/24/12	104.5	25,200	21,420	85	2,772	11	756	3	.....	..	252	1	...	....	2/24/13		
					2/26/12	98.6	8,400	6,300	75	1,680	20	336	4	.....	..	84	1	...	....			
					2/28/12	98.2	8,200	5,576	68	1,968	24	492	6	.....	..	168	2	...	....	.....	.....	Cured.
18	9	Pearl G..	Right lower.....	6	2/20/13	104.6	52,400	40,870	78	7,860	15	2,620	5	1,048	2	...	....	...	....	.....	Urine: alb. trace.	
					2/21/13	104.4	43,600	35,752	82	3,052	7	3,488	8	1,308	3	...	....	...	....			
					2/22/13	101	52,800	42,768	81	4,752	9	4,752	9	528	1	...	....	...	....	2/22/13	Von Pirquet neg.	
					2/26/13	98.6	9,600	6,816	71	2,112	22	576	6	96	1	...	....	...	....	.....	.....	Cured.
					2/28/13	99	7,300	4,526	62	2,190	30	438	6	73	1	73	1	...	....			
19	5	Isadore S.	Right middle lobe only....	3	2/20/13	105	47,000	37,600	80	5,170	11	2,820	6	1,410	3	...	....	...	....	.....	Urine: alb. trace; casts: occasional hyalin.	
					2/21/13	105	33,000	27,060	82	3,960	12	990	3	990	3	...	....	...	....			
					2/23/13	105	20,200	16,160	80	2,828	14	606	3	606	3	...	....	...	....			
					2/24/13	104	19,300	14,475	75	3,860	20	772	4	193	1	...	....	...	....	2/25/13		
					2/26/13	99.6	20,800	15,184	73	3,536	17	1,248	6	624	3	...	....	...	....			
					2/28/13	98.6	16,600	12,616	76	3,320	20	498	3	166	1	...	....	...	....			
					2/30/13	...	9,300	6,882	74	1,953	21	372	4	93	1	...	....	...	....	.....	.....	Cured.
20	5	Max Z...	Right lower. Complication: pleuropneumonia; few c.c. fluid aspirated.	4	1/29/13	103	14,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....	.....	Urine: alb. +; pus 30-40 to field. Pleural fluid P. 87%; S. M. 9%; L. M.	
					1/30/13	104.5	23,000	16,100	70	3,580	16	2,990	13	230	1	...	....	...	....			
					2/ 1/13	103.5	41,200	28,428	69	6,592	16	4,120	10	1,648	4	412	1	...	....			
					2/ 3/13	103.5	43,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....	2/ 3/13		
					2/ 4/13	99	28,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
																					.....	Cured.
21	14	Violet S..	Right upper, middle and lower; left upper and lower. Complications: nephritis; delirium 13 days.	2	10/11/12	105.6	27,600	25,116	91	1,380	5	552	2	552	2	...	....	...	....	.....	Urine: alb. +.	
					10/15/12	101.4	17,500	15,750	90	1,400	8	350	2	.....	..	...	....	...	....	Pseudocrisis		
					10/16/12	106	22,500	19,125	85	2,250	10	900	4	.....	..	225	1	...	....	10/12/13		
					10/17/12	105	21,200	18,868	89	1,696	8	636	3	.....	..	...	...	...	....	10/15/13		
					10/17/12	105.3	21,900	19,710	90	1,533	7	657	3	.....	..	...	....	...	....			
					10/21/12	105.4	34,400	30,960	90	2,752	8	688	2	.....	..	...	....	...	....			
					10/21/12	102.4	31,900	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					10/21/12	105	33,500	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					10/22/12	105	32,800	29,520	90	2,296	7	984	3	.....	..	...	....	...	....			
					10/23/12	104.8	31,600	28,440	90	2,212	7	948	3	.....	..	...	....	...	....			
					10/24/12	98.2	28,000	22,400	80	5,040	18	560	2	.....	..	...	....	...	....	10/23/13		
					10/25/12	99	20,000	14,200	71	4,400	22	1,400	7	.....	..	...	....	...	....			
					10/26/12	99.6	15,700	9,891	63	4,082	26	1,570	10	.....	..	157	1	...	....			
					10/27/12	99.6	15,000	8,700	58	4,350	29	1,800	12	.....	..	150	1	...	....			
					10/28/12	99	15,000	8,100	54	5,850	39	900	6	.....	..	...	1	...	....			
					10/29/12	98.8	12,800	6,656	52	4,480	35	1,536	12	.....	..	128	1	...	....			
					10/30/12	98.6	8,160	3,916.8	48	2,937.6	36	1,305.6	16	.....	..	...	....	...	....			
					10/31/12	...	14,000	7,620	53	5,600	40	980	7	.....	..	...	....	...	....			
					11/ 1/12	...	12,000	5,760	48	5,400	45	840	7	.....	..	...	....	..	....			
					11/ 2/12	...	10,000	6,100	61	3,100	31	600	6	.....	..	200	2	...	....			
					11/ 5/12	...	10,000	5,900	59	3,600	36	400	4	.....	..	100	1	...	....	.....	.....	Cured.

					10/30/12	98.6	8,160	3,916.8	48	2,937.6	36	1,305.6	16	.....	..	...	....	...	....			
					10/31/12	...	14,000	7,620	53	5,600	40	980	7	.....	..	...	....	...	....			
					11/ 1/12	...	12,000	5,760	48	5,400	45	840	7	.....	..	...	....	...	....			
					11/ 2/12	...	10,000	6,100	61	3,100	31	600	6	.....	..	200	2	...	....			
					11/ 5/12	...	10,000	5,900	59	3,600	36	400	4	.....	..	100	1	...	....	.....	.....	Cured.
22	9½	Abe W....	Right upper and middle; left upper and lower. Complication: nephritis after scarlet fever seven years ago.	2	11/ 4/11	105	31,000	27,900	90	2,480	8	620	2	.....	..	...	...	...	....	.....	Urine: alb. trace; casts gran.	
					11/ 5/11	105.6	32,000	28,800	90	2,240	7	960	3	.....	..	...	....	...	....			
					11/ 6/11	104.8	18,400	15,640	85	2,208	12	552	3	.....	..	...	....	...	....			
					11/ 7/11	104	18,600	16,182	87	2,046	11	372	2	.....	..	...	....	...	....			
					11/ 8/11	104	22,000	19,140	87	2,200	10	660	3	.....	..	...	....	...	....			
					11/ 9/11	103	31,200	27,144	87	3,432	11	624	2	.....	..	...	....	...	....	11/ 9/12	Urine: neg.	
					11/10/11	99	22,400	15,680	70	4,480	20	2,240	10	.....	..	...	....	...	....			
					11/11/11	98.6	22,500	16,200	72	4,950	22	1,350	6	.....	..	...	....	...	....			
					11/12/11	99	18,200	12,194	67	3,458	19	2,184	12	.....	..	364	2	...	....			
					11/13/11	99.2	17,000	11,500	68	2,550	15	2,040	12	.....	..	680	4	170	1			
					11/14/11	100.8	14,800	9,620	65	2,960	20	1,480	10	.....	..	592	4	148	1			
					11/15/11	98.4	14,000	8,960	64	3,920	28	1,120	8	.....	..	...	....	...	....			
					11/16/11	98.6	11,800	7,080	60	3,540	30	944	8	.....	..	236	2	...	0			
					11/17/11	98.6	10,300	5,871	57	3,296	32	927	9	.....	..	...	....	206	2			
					11/18/11	98.4	11,200	6,720	60	3,360	30	560	5	.....	..	224	2	336	3			
					11/19/11	98.6	10,500	5,775	55	3,780	36	735	7	.....	..	105	1	105	1			
					11/20/11	98.2	14,300	7,150	50	5,720	40	1,144	8	.....	..	...	....	286	2			
					11/21/11	99	14,300	8,866	62	4,290	30	1,144	8	.....	..	...	....	...	....	... ...	.....	Cured.
23	7	Eva S....	Right upper and middle..	4	6/ 7/11	102	22,000	17,160	78	3,960	18	440	2	.....	..	440	2	...	....	6/ 6/11	Urine: neg.	
24	11	Dora G....	Right upper; left lower..	2	3/ 5/11	105	34,000	26,520	78	2,720	8	4,080	12	340	1	340	1	...	....			
					3/ 7/11	105.5	33,000	28,380	86	1,980	6	1,980	6	660	2							
					3/ 9/11	99	15,800	12,324	78	.....	..	3,160	20	158	1	158	1	...	....	3/ 7/09	.....	Cured.
25	6½	Jenny H..	Right upper and middle..	5	2/ 9/11	104.8	36,900	30,258	82	2,952	8	1,476	4	1,476	4	1,038	2	...	....	2/11/06	.....	Cured.
26	7	Morris E.	Left lower.....	3	2/ 5/11	104	.....	.....	83	.....	4	.....	10	.....	3	...	...	...	....	.....	Urine: alb. +; casts granular.	
					2/ 8/11	98.6	17,200	10,922	63.5	3,440	20	1,290	7.5	688	4	744	4.5	86	5	2/ 6/11		
																...	....	...	....			Cured.
27	12	Louis S...	Right middle and lower..	4	9/14/11	104	20,000	14,400	72	4,200	21	1,200	6	.....	..	200	1	...	....	9/17/11	Urine: alb. +; casts hyaline V.P.+.	
28	4	Lilly F...	Unresolved pneumonia....	..	8/ 8/11	...	13,500	10,935	77	2,430	18	420	4	.....	..	135	1	...	....	Lysis.		Improved.
29	8	John C...	Unresolved pneumonia....	4	5/19/11	...	59,800	49,036	82	8,372	14	2,392	4	.....	..	...	....	...	....	Lysis.		
30	11	Sam G...	Complication: mitral and aortic insufficiency.	...	9/11/11	...	12,000	9,600	80	1,560	13	480	4	120	1	120	1	120	1			
31	12	Ike K....	Right upper and lower..	2	10/18/12,	103	22,800	20,748	91	1,140	5	674	3	.....	..	228	1	...	....	.....	Urine: alb. trace.	
					10/25/12	102.5	18,000	.....	..	.....	..	.....	..	.....	..	...	...	...	....	10/28/12	V.P. neg.	Cured.

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count: Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.	Crisis Date of	Laboratory Examination	Prognosis
47	2	Joe C....	Lobar pn.; right lower. Complication: furunculosis of scalp.	8	3/19/13	105	20,400	16,116	79	3,876	19	408	2	.....	..	...	....	...	....	.....	Urine: alb. trace.	
					3/20/13	105	19,600	16,072	82	2,744	14	392	2	196	1	...	....	...	....			
					3/21/13	103	22,800	18,240	80	3,876	15	456	2	228	1	456	2	...	....			
					3/22/13	103	19,000	15,200	80	3,230	17	190	1	190	1	...	....	190	1			
					3/23/13	102.6	22,400	17,696	79	4,256	19	.....	..	.....	..	224	1	224	1	3/24/13		
					3/24/13	102	24,400	20,008	82	3,416	14	732	3	2	1	...	....	...	....			
					3/25/13	98-101	25,200	19,656	78	4,284	17	504	2	252	1	252	1	252	1			
					3/26/13	100.5	18,000	13,500	75	3,600	20	540	3	180	1	180	1	...	....			
					3/27/13	99	14,400	.....	..	.....	..	.....	..	.....	..	...	....	...	....	.....	.....	Cured.
48	1	Harry G..	Lobar pn.; right upper; left lower. Complication: nephritis, April 12; general edema, April 16; double otitis, April 16; lumbar abscess, May 3.	4	4/ 2/13	104	30,400	24,624	81	4,256	14	1,216	4	.....	..	...	....	304	1			
					4/ 3/13	105	34,200	27,360	80	5,472	16	1,026	3	342	1	...	....	...	....			
					4/ 8/13	104.5	36,400	29,848	82	5,624	16	728	2	.....	..	...	....	...	....			
					4/ 9/13	99	15,400	11,396	74	2,772	18	616	4	308	2	...	....	308	2	4/ 8/09	Urine: alb. + + +;	
					4/10/13	101	18,400	13,248	72	3,312	18	1,104	6	552	3	...	....	184	1	Complication 4/8	Casts, hyaline and granular.	
					4/11/13	103	25,100	18,825	75	4,267	17	1,004	4	502	2	...	....	251	1			
					4/13/13	104	23,000	17,480	76	4,140	18	690	3	460	2	...	....	230	1			
					4/14/13	98	47,000	40,420	86	4,700	10	940	2	940	2	...	....	...	....			
					5/ 4/13	...	15,900	5,088	32	8,268	52	1,590	10	636	4	...	....	318	2	.....	.....	Improved.
49	6	Max B...	Lobar pn.; right upper, middle and lower. Complication: slow resolution, decubitus, furunculosis.	4	11/11/13	105	32,800	26,240	80	3,936	12	2,624	8	.....	..	...	....	...	....	11/17/13	Urine: alb. +.	
					11/19/13	99	28,800	23,040	80	3,744	13	1,728	6	.....	..	...	....	288	1	Pn. unresolved.	casts, hyaline	
																					V.P.—.	Improved.

TABLE 12.—Series 10. Pneumonia with Ascaris Lumbricoides

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count: Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.	Crisis Date of	Laboratory Examination	Prognosis
50	3	Esther L..	Lobar pn.; left upper and lower. Complication: ascaris lumbricoides in stool.	8	2/19/13	105.5	22,000	13,680	62	5,980	27	1,760	8	220	1	440	2	...	....	.....	Urine: alb. ++;casts, hyaline and granular; V. P. neg.	Cured.
					2/20/13	104	18,200	12,740	70	4,368	24	546	3	546	3	...	....	...	....			
					2/21/13	103	36,000	21,040	64	9,720	27	1,800	5	1,080	3	360	1	...	....			
					2/22/13	101	24,000	16,320	68	5,760	24	960	4	720	3	...	...	240	1	2/22/13		
					2/23/13	98	15,200	9,424	62	3,952	26	912	6	608	4	152	1	152	1			

																						tive.	Improved.
																						V.P. positive.	Improved.
41	4	Celia S...	Complication: pleurisy with effusion.	..	10/24/12	...	20,000	14,000	70	3,080	15	1,800	8	1,480	1	...	....	...	....	Lysis.			
42	8	Ida S....	Complication: pleurisy with effusion.	..	8/ 4/12	...	19,900	17,313	87	1,791	9	597	3	199	1	...	....	...	....	Lysis.	V. P. neg.; subcut. tuberculin positive.	Improved.	
43	12	David C..	Complication: pleurisy with effusion.	..	8/ 2/12	...	14,000	12,180	87	.....	..	.....	..	.....	2	...	....	...	....	Lysis.	V.P. positive.	Improved.	
44	8	Bennie L.	Complication: T.B. pleurisy, aspirated.	..	8/23/12	...	11,600	8,236	71	2,320	20	1,044	9	.....	2	...	....	...	....	Lysis.	V.P. positive.	Improved.	

TABLE 10.—Series 8. Tuberculous Pneumonia

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count												Date of Crisis	Laboratory Examination	Prognosis
								Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.			
45	1½	Milton C.	Unresolved pn. left apex..	..	1/18/12	...	20,800	16,688	56	12,516	42	298	1	298	1	...	....	...	....	Lysis.	.....	Improved.
46	1	Alex B...	Both lower lobes.....	..	6/16/12	...	25,600	14,848	58	9,472	37	1,024	4	.....	..	...	....	256	1	.....	.....	Died.

TABLE 11.—Series 9. Pneumonia with Pus Infections

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count												Date of Crisis	Laboratory Examination	Prognosis
								Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.			
					3/19/13	105	20,400	16,116	79	3,876	19	408	2	.....	..	...	....	...	....	.....	Urine: alb. trace.	
					3/20/13	105	19,600	16,072	82	2,744	14	392	2	196	1	...	....	...	....			
					3/21/13	103	22,800	18,240	80	3,876	15	456	2	228	1	456	2	...	....			
					3/22/13	103	19,200	15,360	80	3,230	17	190	1	190	1	...	....	190	1			
					3/23/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	224	1	224	1			
					3/24/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....	3/24/13		
					3/25/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					3/26/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					3/27/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					3/28/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					3/29/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					3/30/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					3/31/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/ 1/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/ 2/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/ 3/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/ 4/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/ 5/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/ 6/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/ 7/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/ 8/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/ 9/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/10/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/11/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/12/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/13/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/14/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/15/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/16/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/17/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/18/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/19/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/20/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/21/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/22/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/23/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/24/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/25/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/26/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/27/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/28/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/29/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					4/30/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/ 1/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/ 2/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/ 3/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/ 4/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/ 5/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/ 6/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/ 7/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/ 8/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/ 9/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/10/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/11/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/12/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/13/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/14/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/15/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/16/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/17/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/18/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/19/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/20/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/21/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/22/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/23/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/24/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/25/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/26/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/27/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/28/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/29/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/30/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					5/31/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/ 1/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/ 2/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/ 3/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/ 4/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/ 5/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/ 6/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/ 7/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/ 8/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/ 9/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/10/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/11/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/12/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/13/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/14/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/15/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/16/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/17/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/18/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/19/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/20/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/21/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/22/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/23/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/24/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/25/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/26/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/27/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/28/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/29/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					6/30/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/ 1/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/ 2/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/ 3/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/ 4/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/ 5/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/ 6/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/ 7/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/ 8/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/ 9/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/10/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/11/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/12/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/13/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/14/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/15/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/16/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/17/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/18/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/19/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/20/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/21/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/22/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/23/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/24/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/25/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/26/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/27/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/28/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/29/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/30/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					7/31/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/ 1/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/ 2/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/ 3/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/ 4/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/ 5/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/ 6/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/ 7/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/ 8/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/ 9/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/10/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/11/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/12/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/13/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/14/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/15/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/16/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/17/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/18/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/19/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/20/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/21/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/22/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/23/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/24/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/25/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/26/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/27/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/28/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/29/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/30/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					8/31/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/ 1/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/ 2/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/ 3/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/ 4/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/ 5/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/ 6/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/ 7/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/ 8/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/ 9/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/10/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/11/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/12/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/13/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/14/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/15/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/16/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/17/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/18/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/19/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/20/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/21/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/22/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/23/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/24/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/25/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/26/13	103	18,000	14,400	80	3,060	17	180	1	.....	..	...	....	...	....			
					9/27/13	103	18,000	14,400	80	3,

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count: Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.	Date of Crisis	Laboratory Examination	Prognosis
36	3	Winifred H.	Right upper. Complication: empyema, March 10, 1913; aspirated March 10, 1913; operated March 18, 1913.	5	3/ 5/13	104	19,300	16,791	87	1,737	9	579	3	193	1	...	....	...	....	.....	Urine: trace alb. V.P. neg.	
					3/ 6/13	104	19,500	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					3/ 8/13	105.5	28,600	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					3/10/13	104.8	40,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					3/12/13	103	48,400	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					3/14/13	105.4	43,800	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					4/12/13	105.4	33,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....	.....	.....	Died.
37	10*	Morris A.	Complication: empyema...	..	7/ 5/12	...	58,300	44,308	76	13,409	23	583	1	.....	..	...	....	...	....	Lysis.	Von Pirquet neg.	Improved.
38	12½	Ratner W.	Complication: empyema, bilateral.	..	10/24/12	...	31,500	26,145	83	3,780	12	1,575	5	.....	..	...	....	...	....	Lysis.	Cultures and V. P. neg.	Cured.
39	3½	Hyman S.	Complication: empyema...	..	12/31/12	...	45,000	38,250	85	4,500	10	2,250	5	.....	..	...	....	...	....	.....	V.P. and cult. of fluid neg.	

* Ten weeks.

TABLE 9.—Series 7. Lobar Pneumonia with Pleurisy

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count: Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.	Date of Crisis	Laboratory Examination	Prognosis
40	12	Ike K....	Whole left and right upper. Complication: pleurisy with moderate effusion; not operated.	2	10/19/12	103	22,800	20,748	91	1,568	5	684	3	228	1	...	....	...	....	.....	Urine: alb. trace; V. P. neg.	
					10/25/12	102	18,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					10/29/12	99	24,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....	Lysis.		Cured.
41	4	Celia S...	Complication: pleurisy with effusion.	..	10/24/12	...	20,000	14,000	70	3,000	15	1,600	8	1,400	7	...	....	...	....	Lysis.	V.P. positive.	Improved.
42	8	Ida S....	Complication: pleurisy with effusion.	..	8/ 4/12	...	19,900	17,313	87	1,791	9	597	3	199	1	...	....	...	....	Lysis.	V.P. positive.	Improved.
43	12	David C..	Complication: pleurisy with effusion.	..	8/ 2/12	...	14,000	12,180	87	.....	..	.....	..	.....	..	...	....	...	....	Lysis.	V. P. neg.; subcut. tuberculin positive.	Improved.
44	8	Bennie L.	Complication: T.B. pleurisy, aspirated.	..	8/23/12	...	11,600	8,236	71	2,320	20	1,044	9	.....	2	...	....	...	....	Lysis.	V.P. positive.	Improved.

TABLE 10.—Series 8. Tuberculous Pneumonia

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count: Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.	Date of Crisis	Laboratory Examination	Prognosis
45	1½	Milton C.	Unresolved pn. left apex..	..	1/18/12	...	29,800	16,688	56	12,516	42	298	1	298	1	...	....	...	....	Lysis.	.....	Improved.
46	1	Alex B...	Both lower lobes.....	..	6/16/12	...	25,600	14,848	58	9,472	37	1,024	4	.....	..	...	....	256	1	.....	.....	Died.

TABLE 11.—Series 9. Pneumonia with Pus Infections

TABLE 7.—SERIES 5. BRONCHOPNEUMONIA

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count: Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.	Date of Crisis	Laboratory Examination	Prognosis
32	½	Morris G.	Diffuse, both lungs. Complication: endocarditis (breast fed).	5	2/23/13	103	19,400	7,566	39	11,058	57	388	2	388	2	...	....	...	....			
					2/24/13	105.5	43,600	18,312	42	21,800	50	2,616	6	872	2	...	....	...	....			
					2/25/13	103	32,000	15.040	47	13,760	43	1,600	5	1,280	4	...	....	320	1	.....	.....	Died.
33	5	Harry W..	Both lower lobes irregularly and diffusely involved, prob. influenzal pneumonia.	19	1/10/13	102	12,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....	.....	Urine: alb. trace; casts; hyalin; diazo neg. V.P. neg.	
					1/11/13	102.5	7,500	3,375	45	3,750	50	300	4	75	1	...	....	...	....			
					1/12/13	101	5,600	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					1/16/13	104.5	10,000	.....	..	.....	..	.....	..	.....	..	...	....	...	....	Lysis.		
					1/21/13	102.5	9,800	.....	..	.....	..	.....	..	.....	..	...	....	...	....			
					1/27/13	100.5	14,206	.....	..	.....	..	.....	..	.....	..	...	....	...	....	.....		
					2/19/13	101.2	25,000	17,500	70	5,500	22	750	3	750	3	500	2	...	....	.....	.....	Cured.
34	½	Leslie M..	Diffuse, both lungs. Complication: myocarditis, rachitis, nephritis (breast fed).	14	2/17/13	104.5	24,000	12,960	54	8,160	34	1,440	6	480	2	960	4	...	....	.....	Urine: alb. +; casts: hyaline and granular.	
					2/18/13	106.5	12,300	6,888	56	3,690	30	861	7	492	4	246	2	123	1			
					2/19/13	104	20,600	10,094	49	7,416	36	1,854	9	1,030	5	206	1	...	....			
					2/20/13	102	32,800	19,680	60	9,840	30	2,296	7	984	3	...	....	...	....			
					2/21/13	102	24,200	12,584	52	6,776	28	1,936	8	2,904	12	...	....	...	....			
					2/22/13	105	25,300	13,662	54	8,076	32	2,024	8	1,518	6	...	....	...	....			
					2/23/13	102	42,000	27,720	66	10,080	24	2,940	7	1,260	3	...	....	...	....	.....	.....	Died.
35	2/12	Baby Nixon..	Left upper and lower. Complications: fibrinous pleurisy, purulent meningitis, tetany, rickets.	2	2/21/13	103	56,200	35,406	63	10,678	19	6,744	12	3,372	6	...	....	...	....	.....	Urine: alb. trace. Aspirated fluid; pneumococci but no pus. V. P. neg.	Died.

TABLE 8.—SERIES 6. LOBAR PNEUMONIA WITH EMPYEMA

Case No.	Age Years	Name	Diagnosis and Complications	Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential White Count: Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent.	Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.	Date of Crisis	Laboratory Examination	Prognosis
36	2	Ruth S..	Left upper and lower. Complications: empyema, pericarditis, endocarditis.	5	2/23/13	103	19,400	7,566	39	11,058	57	388	2	388	2	...	....	...	....			
					2/24/13	105.5	43,600	18,312	42	21,800	50	2,616	6	872	2	...	....	...	....			
					2/25/13	103	32,000	15.040	47	13,760	43	1,600	5	1,280	4	...	....	320	1	.....	.....	Died.
37	3	Ethel M..	Left upper and lower. Complications: empyema, pericarditis, endocarditis.	5	2/23/13	103	19,400	7,566	39	11,058	57	388	2	388	2	...	....	...	....			
					2/24/13	105.5	43,600	18,312	42	21,800	50	2,616	6	872	2	...	....	...	....			
					2/25/13	103	32,000	15.040	47	13,760	43	1,600	5	1,280	4	...	....	320	1	.....	.....	Died.

parallel to the polymorphonuclear neutrophils and there is often an absolute increase and they remain high even after the crisis. Myelocytes and megaloblasts may occur in severe cases.

It is of interest to note that a peculiar relation seems to exist between the appearance in the blood and tissues of lymphocytes and eosinophil cells; neither are apt to appear when the neutrophilic cells constitute the prevailing cell in the blood or exudate, but either may be found in the less acute exudative processes. The sequence of lymphocytosis followed by eosinophilia is especially clear after the injection of tuberculin (Grawitz), and after splenectomy in animals (Kurloff). In the majority of cases reported as showing lymphocytosis no distinction is made between lymphocytes and large mononuclear leukocytes, yet there is considerable evidence to show, as Erhlich believes, that these cells represent a separate theory and in some conditions the large mononuclear leukocytes and not the lymphocytes are increased. In Von Jaksch's anemia, rachitis (Rieder), syphilis (Rille), scarlet fever and small-pox and acute leukemia (Frankel), the significance of the increase of the large mononuclear cells, apart from the lymphocytes, cannot at present be stated.

CONCLUSIONS AS TO PNEUMONIA

1. Leukocytosis in pneumonia as in other infections is dependent on two great factors: first, the reaction on the part of the organism, and second, the severity of the infection. This is peculiarly true of children whose course in the typical types is associated with a lower mortality than in adults while in the atypical, secondary types, we see the highest mortality of any age except that of senility.

2. Generally speaking, a high count indicates a severe infection in an individual of strong powers, a moderate increase indicates either a slight infection with good resistance, or a severe type with an inadequate reaction. A low count suggests either a slight infection or an overpowering influence.

3. It is impossible to make a reliable prognosis in pneumonia from the leukocyte count alone; however, when studied in relation to the clinical picture, the leukocyte count becomes of great prognostic significance.

4. Primary pneumonias of infancy are usually of the lobar type. Bronchopneumonias are usually either secondary pneumonias or are seen in infants with low vitality. This assertion is based on the physical findings, clinical course and blood-findings.

5. Young infants react to infections with a high neutrophil count, the same as seen in older children.

6. The usual blood-picture in pneumococcus infections is that of a high neutrophil count with a moderate lymphocytosis before the crisis. A deviation from this picture should lead one to suspect lues, tuber-

culosis, typhoid or influenzal or other infections, or the presence of a rachitis or other complication. Excessively high counts are apparently more prone to the development of empyema and other complications, although it is impossible to say whether or not the hyperleukocytosis is not the result of an already infected pleura, etc., not demonstrable clinically. In several instances the toxic symptoms associated with these high counts have led us to do spinal puncture with negative findings.

7. The neutrophil and lymphocyte curves approximate and occasionally cross each other during convalescence. There is a rapid fall in the neutrophils after the crisis.

8. Pseudocrisis is associated with little or no reduction in the total count.

9. A slow fall in the leukocytes with continued high fever indicates either delayed resolution or complications. This is commonly seen in the secondary pneumonias.

10. Sudden increases in the total and neutrophil counts during convalescence should lead to the suspicion of complications, as should a tendency to remain high.

11. In secondary pneumonias the count varies with the associated complications. Frequently there is a high lymphocyte count.

12. Eosinophilia:

1. Eosinophils practically disappear during the height of the disease and usually reappear just before or following the crisis or with the beginning of lysis. Their recurrence can usually be considered a good sign.

2. Disappearance of eosinophils does not depend on the presence or absence of a leukocytosis. When they have disappeared, however, the absolute decrease tends to remain until the white count nears the normal.

3. They rarely reach an absolute count much above the normal.

4. Their occurrence usually indicates that the infection has passed its acme, and is therefore of practical value for prognosis.

5. Their disappearance may be retarded by outside factors, as the presence of ascaris lumbricoides in the intestinal tract in Case 50 and repeated pneumococcus infections in Case 13.

6. Their disappearance following their return with beginning convalescence should lead to the suspicion of beginning complications.

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DISSEMINATED MILIARY TUBERCULOSIS OF LUNGS AND SKIN *

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The diagnosis of miliary tuberculosis in the very young is difficult. In the lungs, for instance, without consolidation and without cavity, miliary tuberculosis may be strongly suspected, but not easily proved.

I submit herewith two helps to the diagnosis — one, of the lungs — the x-ray; the other of the skin — the eruption. The first case selected for illustration shows both diagnostic signs and will be submitted complete. The best recent x-ray apparatus has made it possible to produce the accompanying pictures, which speak for themselves. In six cases of infants and young children suspected of disseminated miliary tuberculosis of the lungs, the x-ray has cooperated with other tests to decide the diagnosis. For illustration of what can be done, the best x-ray plate has been selected, the best print made from it and the best result obtainable here presented (Fig. 1).

The second help to the diagnosis of general miliary tuberculosis in the young is general miliary tuberculosis of the skin. The localized lesions of the skin so often described, are not here meant. (See Figs. 2, 3 and 4.)

CASE 1. General miliary tuberculosis; involvement of skin.

Dorothy K., 2½ years old, was admitted to the Presbyterian Hospital with the diagnosis of pneumonia. The case presents the following points of interest: 1. The patient had the appearance and the chart of a child sick with pneumonia but with the physical signs of bronchitis. 2. The exact diagnosis was made by x-ray (lungs). (Fig. 5). 3. There was the unusual skin eruption of disseminated miliary tuberculosis. (Figs. 2, 3, 4.) 4. A necropsy completed the record and confirmed all previous findings.

1. Physical signs: As to the "look and chart" of pneumonia in this child, the temperature (T) was high and continuous, 102 to 105 F.; the pulse (P) was 130 to 160 per minute; the respiration (R) was 42 to 72; out of normal proportion to the pulse; the R-P ratio was "disturbed." "When the R-P ratio departs from the normal of ¼ and approximates 1/3, look for pneumonia" (O'Dwyer). It is to children that I would apply this; it is not so valuable in adults.

It would be exceptional for a pneumonia patient to remain in hospital a week developing a severe febrile reaction, without revealing some signs of consolidation. Presumably all clinical men will think there is here shown good reason for the diagnosis of tuberculosis. That is granted. For exact diagnosis by means of new and high grade mechanical appliances, however, we may still contend.

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* Read before the Association of American Physicians, Washington, D. C., May 7, 1913.

2. Exact Diagnosis by X-ray: At the last meeting of this Association I presented in discussion of Dr. Sewell's paper an x-ray plate showing clearly disseminated miliary tuberculosis of the lungs without consolidation; also without excavation. (Fig. 1.) At this time I am prepared to submit the x-ray plates of six cases including the first shown. Two of the cases have been confirmed by necropsy (the one here shown being included), one has been corroborated by inoculation into the guinea-pig, one was confirmed by examination of an excised gland; others by finding bacilli in the throat swabs, etc., till we can be rationally certain that our x-ray findings are reliable and trustworthy.

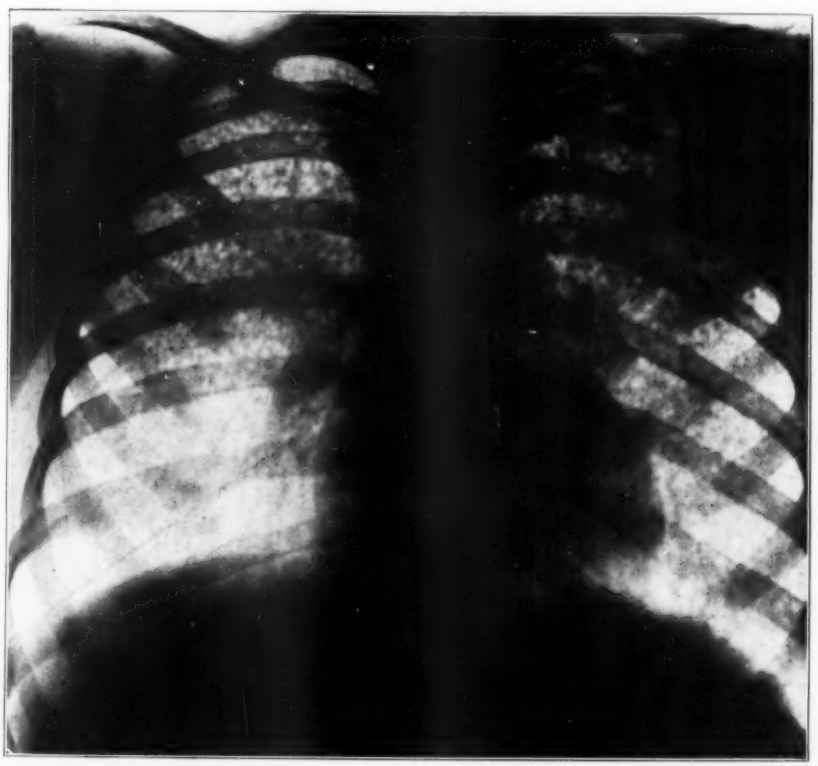

Fig. 1.—General miliary tuberculosis. Radiograph of thorax of Rosie R., aged 8 years. Tubercle bacilli recovered from the spinal fluid. No necropsy.

3. Skin Eruption: Miliary skin tuberculosis, necrotic skin tuberculid—the names suggested are sufficiently descriptive. This is an unusual though not rare lesion. In the Presbyterian Hospital three cases have been seen. I personally have seen but two. (Figs. 2, 3, 4.)

The character of the eruption is papulo-vesicular, perhaps more vesicular from the first. On the neck, in the above case, the vesicles were more like sudamina, a little larger, though not as large as the vesicle of chicken-pox. They were thickly clustered in the neck. Later there developed a hemorrhagic zone about the vesicle and a subsequent drying and crusting in the middle of the lesion. The completed individual lesion was about 2 mm. in diameter. This stage is figured in Pfaundler and Schlossmann in an excellent picture to which the reader is referred. The illustrations are colored and are the only ones

known to us. The accompanying photographs of the present case, in black and white, show to good advantage. (Figs. 2, 3, 4.) The rash was on all skin surfaces, including soles, palms and scalp, with more about the mouth, anus and buttocks. The superficial lymph-nodes were enlarged everywhere; the liver was enlarged. Naturally the case attracted a good deal of attention. Colleagues who were called in to see the case expressed their views, one suggesting syphilis, another chicken-pox. The rash is said to have been evident three days before entrance, appearing first about the mouth, neck and chest.

4. Necropsy: The brain was not examined; tubercles were found in the lungs, spleen, kidneys, liver, etc. Tubercle bacilli were found in clumps in the blood of the heart in any casual smear. Smears taken from the skin vesicles showed such quantities that the spread-out corium was faintly pink from the stained bacilli. The case was acute, which may have determined the character of lesions.

It is interesting to note that the structure of the tubercles was not the usual structure; in fact, strictly speaking, they were not tubercles at all. More

Fig. 2.—Miliary tuberculosis of the skin. Papulo-vesicles; hemorrhagic crusts (umbilicated). Properly, necrotic tuberculid.

properly speaking, the lesion was necrotic tuberculosis ("tuberculid"); there was no tissue reaction at all, merely melting down into necrosis where tubercle bacilli stopped and began to grow. There was no connective tissue reaction, no giant cells, no surrounding zone of epithelioid and mononuclear wandering cells. "There were just necrotic areas filled with tubercle bacilli. In none of the zones were found evidences of protective reaction" (Dr. Lamb). Simply larger and smaller areas of necrosis, lymph-nodes and tonsils containing the same.

Six cases are shown in the x-ray plates. All of the patients came into the hospital with the evidence in favor of acute pneumonia, the chart bearing out the general suggestion and appearance of the disease. The x-ray was really the first means of positive diagnosis, being subsequently confirmed by finding tubercle bacilli.

Tuberculous eruption on the skin, necrotic tuberculid, disseminated miliary tuberculosis of the skin, suggest in their names the characteristics of the lesions under consideration.

Case 2.—General miliary tuberculosis involving the skin in a child aged 18 months. Died. No autopsy.

Kate N. entered the Presbyterian Hospital with the diagnosis of pneumonia. The onset was abrupt three days before, with drowsiness, fever, cough and a peculiar, unrecognized rash.

Examination showed a temperature of 101 to 103 F., rising to 105.5 F. at or just before death, cough, coarse râles over both lungs, harsh breathing and voice over irregular areas, rest'essness, flaring nostrils—satisfactory evidences

Fig. 3.—Tuberculosis of skin; papulo-vesicular; crusts (umbilicated); hemorrhagic. Properly, necrotic tuberculid.

of pneumonia. As the Roentgen-ray was not in common use at that time, no radiographs can be shown. There was no autopsy. No bacilli were found.

Skin Eruption: In order of time, this case was the first case that had come under the observation of the staff and was not recognized until an excellent colored illustration in Pfaundler and Schlossmann was noticed. It is important to have this case in mind in deciding on skin eruptions, for those who see one case and recognize its character will never make a mistake. Witness the fact that the two cases accompanying this were recognized instantly by those who saw the first, and by no others present.

The eruption was discrete, papulo-vesicular, later drying, "topped with a scab," about 2 mm. in diameter, and, as the recorder says, "suggestive of chicken-pox, except that the papules are small."

An expert from the Board of Health pronounced it a late stage of chicken-pox, thus explaining why they were so small.

The lesion is called by Pfaundler and Schlossmann "necrotic tuberculid."

CASE 3.—Pulmonary tuberculosis and skin tuberculids.

John H., 15 months old. This patient also came to hospital with the diagnosis of acute pneumonia. The patient died; no necropsy and no radiographs made, but the tissues of the skin lesion were inoculated into guinea-pigs and proved to be tuberculosis.

The patient was in hospital five days, in a dying condition all the time. The lungs showed râles everywhere, bronchial voice and breathing, harsh in nature.

The main interest centers about the skin lesion, small pin-head-sized and a little larger papules with small necrotic centers. There were a few on the arms and legs, a few on the back and front of the trunk, also one or two on the thighs. There were a few small ecchymotic spots on the thighs. The diagnosis was pulmonary tuberculosis, and on the skin, tuberculids. When engrafted in the guinea-pig, typical tuberculosis in the internal organs was developed.

Fig. 4.—Miliary tuberculosis of skin; papulo-vesicular; hemorrhagic; crusts (umbilicated). Properly, necrotic tuberculid.

CASE 4.—General miliary tuberculosis of lungs; x-ray; autopsy.

William F. (colored), aged 10 months, entered the hospital with the diagnosis of pneumonia. There were fever, apathy ("dopey"), R-P ration disturbed, 1-3; respiration, 36 to 50 per minute; pulse, 110 to 150 per minute, irregular; temperature, 100.5 to 102.5 F.; discharging ear; vomiting—manifestly a very sick child; no consolidation of the lungs, no tubercle bacilli found, no history of râles in the chest. The diagnosis was suspected from the history and chart but not confirmed till the x-ray showed it clearly and unmistakably. This was one of the best proofs of the value of the x-ray. As to râles, they were probably to be found at favorable moments, after a long interval of uninterrupted quiet. The hospital intern states that the lungs were clear in front and behind. Spinal fluid was perfectly clear. Noguchi-Wassermann test of blood and spinal fluid was negative.

This case, also entered as one of pneumonia, was then believed to be tuberculosis from the history and chart, and the diagnosis was confirmed by the x-ray and at last by autopsy. The radiograph was one of the best of the series.

CASE 5.—Diagnosis on admission, acute tuberculous broncho-pneumonia. General tuberculosis. Died. No necropsy.

Vieto S., aged 3 years. This case is one in which there was good ground for believing that there was tuberculosis, and the x-ray was convincing; there was also confirmation later from the tissue of a superficial gland.

CASE 6.—Diagnosis on admission, general bronchitis, probably tuberculous. X-ray convincing. No corroboration. No necropsy.

Dan B., 6 years old. This case on admission was one that suggested pneumonia, general bronchitis, and at length the conviction that it was probably tuberculosis. The point of interest was the convincing x-ray picture. There was no corroborating evidence of tuberculosis, but the x-ray was so good that there could be no feeling of doubt.

The child had had measles, whooping-cough and pneumonia. He entered the hospital with a cough which had been active for two weeks, and there was evidence of an acute fever-chill and fever, no expectoration, no vomiting. Patient had been treated for malaria. In the lungs were fine, scattered, "squeaking" râles.

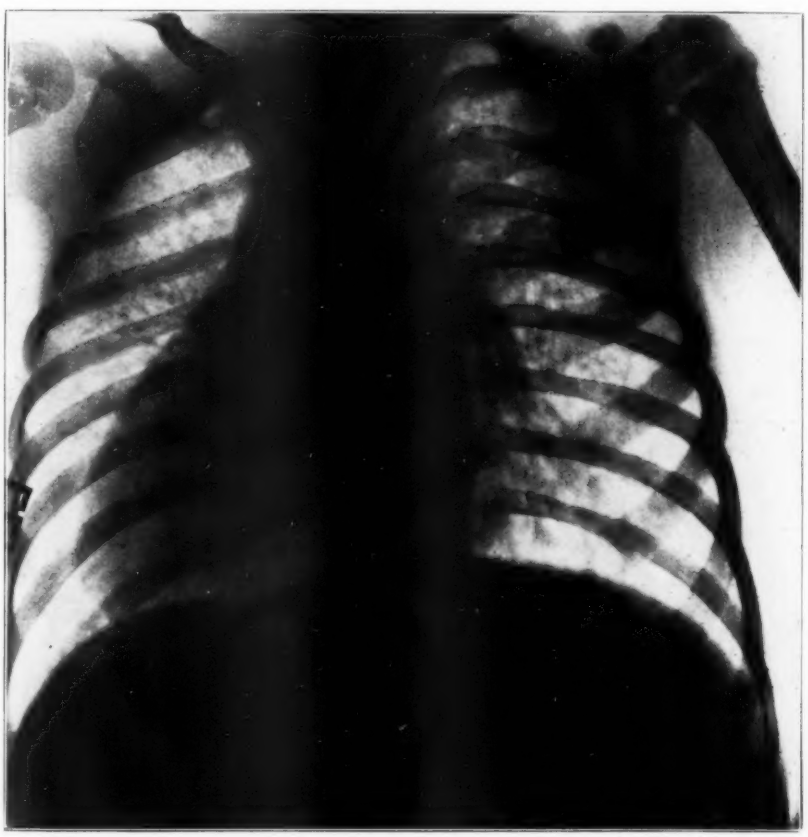

Fig. 5.—Miliary tuberculosis of lungs. Same patient had also miliary tuberculosis of skin (necrotic tuberculid).

There were thought to be certain localities in which the breathing sounds indicated a moderate condensation of the lungs, but the x-ray did not corroborate it. There was no record of positive evidence of consolidation. The patient was several weeks in hospital, and the x-ray was the only convincing testimony outside of the clinical.

Case 7.—Entered as pneumonia. Diagnosis by x-ray. Tubercle bacilli found in spinal fluid. No necropsy.

Rosie R. This 8-year-old child was in the hospital twenty-five days, running the course of an old-fashioned consumptive. The history on admission, though,

suggested pneumonia-cough, expectoration, blood-stained sticky expectoration, stabbing pains in the chest, night-sweats and loss of weight.

The previous history was that of measles at 2 years, bronchitis at 7. The interest in the case centers in the x-ray testimony. It was a convincing picture and was corroborated by finding tubercle bacilli in the spinal fluid. (Fig. 1.)

CASE 8.—Diagnosis on discharge was general miliary tuberculosis of the lungs. X-ray. Improved.

John S., aged 9. The interest in this case rests on these points: A suggestive previous history; a positive x-ray picture; "tubercles on the lungs"; good general condition; discharged having no fever and apparently thriving fairly. Sent to the country for still further improvement.

Previous History.—No significant family history or surroundings. Previous illnesses, whooping-cough, measles, and indefinite history of tumors which seem to have been tuberculosis of the lymph-nodes just over sternum and neck. This patient was observed in good hospitals just before coming to the present one, and nothing special was discovered in the lungs. In one particularly good hospital, where the facilities for x-ray would probably be excellent, nothing was sought by x-ray; at least there was nothing said about it.

Examination.—"Bright boy; does not look ill; ears and cheeks bluish-red; not dyspneic; slightly cyanotic; mentally alert; no noteworthy scars; tongue clean in the middle line; lips very red; cervical and inguinal glands enlarged; lungs and abdomen negative; extremities and hands bluish; veins of neck enlarged; a few moist râles, most of which clear up on deep breathing; while in hospital, up and about, comfortable, always cyanosed, notably about the ears and lips, also finger-nails. Fluoroscopic examination confirms x-ray plates described above. Patient went to Loomis Sanatorium. Father reports glands are smaller. Clinical diagnosis, cervical tuberculous adenitis.

DISCUSSION

There is still an added interest to this case. The human interest in a case that by all laws and experience is doomed, yet has gone out of the hospital with lungs thickly studded with miliary tubercles. To be sure, without the recent new appliances above alluded to, the diagnostician might have thought he had made a mistake; the case then would have passed as undetermined and the memory need not concern itself trying to learn anything from it. Whether the further history of this case is ever written cannot now be said. This may be the final outcome — by the use of the x-ray many such histories may accumulate and the profession learn that they have for years been overlooking cases of disseminated miliary tuberculosis of the lung. What the Wassermann reaction is to syphilis, the x-ray promises to be to the diagnosis of lung tuberculosis in children.

These three cases of skin tuberculosis present the characteristic features thus far met in our experience and most of those not recorded by Tileston in his larger experience.

The most profitable impression to carry away from this reading is that the cases present to the experienced observer the mental picture of pneumonia, but the physical signs of bronchitis. At this point the physician may be unable to procure any reliable testimony for exact diagnosis;

and until a good x-ray picture has been obtained he may but conjecture, and he may never know whether or not he has made the correct diagnosis. Next to an autopsy the x-ray gives the most intimate knowledge of the lesion of the child's lung.

Skin eruptions in the feeble and non-resistant are not rare, but are mostly overlooked. The lesion is mainly necrotic, resulting in vesicles. There may be a stage of papulo-vesicle preceding this. The cases here cited are distinctly acute. Our experience leads us to prefer the term given to the lesion by the authors, Pfaundler and Schlossmann, "necrotic tuberculid."

"The fatal result in infants is probably only another manifestation of the more pernicious course of tuberculosis in general in the first years of life."[1]

Briefly stated, the modern x-ray apparatus, next to an autopsy, furnishes the most valuable insight to the lung lesion of infants and young children.

Eruption of miliary tubercles furnishes a pathognomonic sign for diagnosis and a valuable, almost conclusive, point for prognosis; that is, the lesion is nearly always associated with a fatal form of tuberculosis in the young child.

The histology varies with the acuteness of the invasion. The above cases were of the acute type and the vesicles (necroses) were the first revealed lesion. It may be that slower developing lesions would disclose the different types described, viz., "papulo-squamous" and "papulo-necrotic" tuberculids, described by Darier and Boeck in 1896.

Bacilli are found frequently in the blood and the eruption and the lesion is probably always embolic.

Among the characteristics of the individual lesions may be mentioned the *size*, that of a rose spot in typhoid fever, topped by a tiny *vesicle*, surrounded sooner or later with a congested or hemorrhagic *zone*, with the formation of a *crust*, which, when removed, leaves a little *pit*.

The skin lesion is to be differentiated from that of chicken-pox, moluscum contagiosum and from syphilis.

Experience with this eruption is like that with scurvy in infants—once seen the lesion cannot be again overlooked.

57 East Seventy-Ninth Street.

1. Tileston: Arch. Int. Med., July, 1909, iv, 21.

A CONSIDERATION OF TARDY SYPHILIS*

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During the past two years, since the introduction of salvarsan in this clinic, more than one hundred and fifty luetic infants and children have been observed, and over seventy treated with salvarsan.

It is not the object of this paper to give merely a description of the various cases, many of them most interesting, but to discuss two classes of cases, with our results and conclusions. The first group is composed of patients with marked congenital lues, having positive family histories, clinical symptoms and triple + Wassermann reactions. The second group is composed of what we consider the marasmic type of luetic infant, as well as children from a year to sixteen years, with late luetic manifestations.

In the first, or active group, five typical cases, all under 3 months of age, were observed and treated with salvarsan, four directly and one indirectly. All had positive family histories, triple + serum reports and marked clinical evidences of lues. Three of the cases exhibited snuffles, fissured mouths and ani, and marked maculopapular eruptions; one child's mouth was a mass of inflammatory areas and patches, and had to be fed by gavage (Figs. 1, 2 and 3). These cases were given 1/20 gm. of salvarsan intravenously, and all died within two weeks after treatment. It was of interest to note the rapidity with which the skin symptoms cleared up in each case, and with this a general improvement for a few days in the infants' condition. Later, however, they all developed a generalized edema, with slight urinary signs, and died.

The fourth infant, 4 days old, showed a marked syphilitic pemphigus, covering almost the entire body, and died forty-eight hours after the administration of salvarsan. The last case in this group was a luetic mother with an infected infant 8 weeks old. Two weeks after the child was born, the mother broke out with patches in her mouth and a secondary rash. Six weeks later she came to the clinic with the infant, whose body was covered with a bright red rash. The mother's blood and breast-milk showed a positive serum reaction, and she was given an intravenous injection of 6/10 gm. of salvarsan. For the next three days the child

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* Read by invitation before the Alameda County Medical Society, September, 1913.

was nursed every three hours by the mother, and on the second day the rash was more pronounced, which no doubt was a Herxheimer reaction described by Ehrlich. Unfortunately, the mother did not return to the clinic after the third day and the case was lost sight of.

Coming now to the latent group, or what we might designate as tardy syphilis, we wish to consider two classes of cases, the first a class which fills probably one-third of our clinics, namely, syphilitic children showing late manifestations of the disease, in the eyes, ears, bones, nervous

Fig. 1.—Baby M., aged 3 months. Admitted Jan. 2, 1913. Showing eruption on admittance and day salvarsan was injected.

Fig. 2.—Same as Fig. 1.

system, skin, etc., with a positive or negative Wassermann reaction, no clinical history or evidence of lues in infancy, and in many cases with no family history of lues obtainable. The second class comprises a group of infants which simulate in every respect the marasmic type of infant, with the history that, although the child is 3 or 4 months old, its weight has never been above its birth weight, it looks dried out and ill-nourished, has no clinical evidence of syphilis, may or may not have an

obtainable family history, and has a triple + or slightly positive Wassermann. We believe that there is a certain class of marasmic infants who, although they show no clinical evidence of syphilis during early infancy, develop manifestations of the disease in later infancy and childhood, as shown in the first class of cases in this latent group.

Of the series of twenty-three cases reported a year ago,[1] and closely watched since, twenty-one presented late manifestations of lues, and ranged in age from 3 weeks to 16 years. They were divided as follows:

12 cases exhibiting bilateral interstitial keratitis.
2 cases exhibiting bone lesions.
3 cases exhibiting 1. Atrophic rhinitis: 2. Arthritis; 3. Backward growth. 4. Hutchinson teeth and positive Wassermann reaction.

Fig. 3.—January 10. Showing improvement in eruption eight days after injection.

1 case exhibiting encephalitis and interstitial keratitis.
1 case exhibiting chorea.
2 infants exhibiting early congenital symptoms.

Since these cases were reported, many more patients have been observed and treated, but the above are sufficint to bring out the points in this paper.

In the twenty-one cases, a careful history was taken in each, and in only two cases was evidence of early luetic manifestation obtained. This was delayed walking, which is not necessarily an evidence of syphilis. A definite family history was obtained in seven cases.

As regards the Wassermann reaction, all showed a triple + result, with the exception of five of the eye cases; one of the latter became positive two weeks after the third injection. Possibly the four remain-

1. Yerington, H. H.: A Preliminary Report on Twenty-Three Children Treated with Salvarsan. Jour. Cal. St. Med. Soc., Oct. 12.

ing cases were tuberculous in origin, although no other evidences of tuberculosis could be found. As to the results of treatment, we found that four out of the twelve eye cases cleared up, and for six months have shown negative Wassermann reactions; these were cases in which

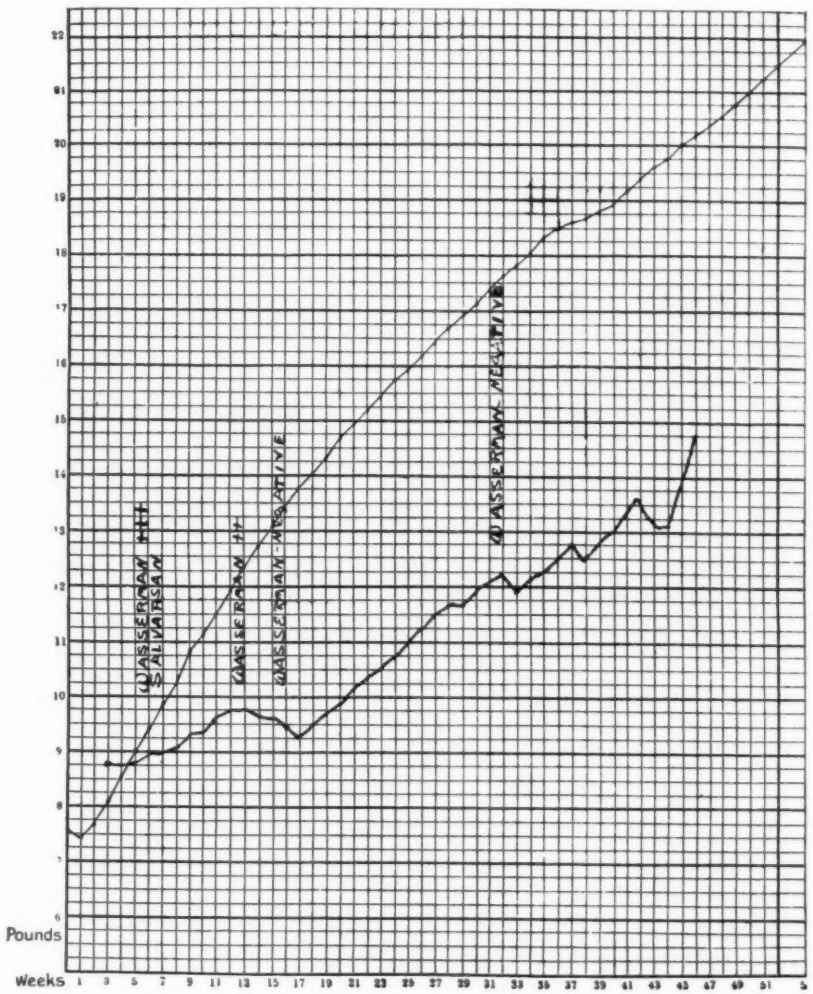

Fig. 4.—Born Aug. 12, 1912. Marasmic type. No clinical luetic symptoms. Salvarsan sixth week. Gain in weight after treatment. Wassermann negative on eleventh and thirty-third weeks.

the duration of the infection was short. Cases of long duration, with repeated attacks, and the four negative cases showed little improvement.

Good results were obtained in bone lesions, both in infants and older children.

The cases of partial deafness, with bilateral keratitis, showed no improvement in hearing.

In the case of chorea, two injections were given in four weeks, and the improvement was more rapid than in the usual case, the Wassermann reaction remaining negative after the second month.

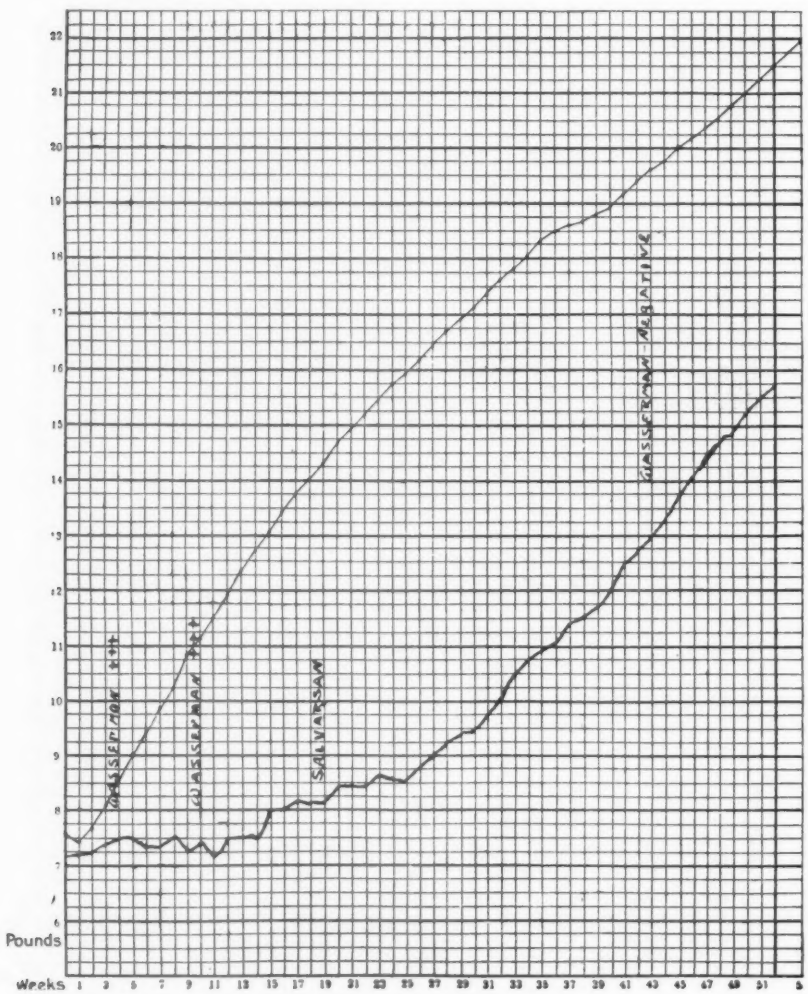

Fig. 5.—A. S., born May 12, 1912. Marasmic type. No clinical luetic symptoms. Salvarsan nineteenth week. Steady gain following treatment, with a negative Wassermann on thirty-third week.

The case of encephalitis with keratitis did not improve, although a negative Wassermann reaction was obtained after the third injection. We found, however, that in this case and others exhibiting Hutchinson's teeth, backward growth, and anemia, an improvement in the general

condition of the patients occurred, which was due no doubt to the tonic effect of the drug.

It is our opinion that in order to obtain good results in such a group of late congenital lues, at least three injections must be given at frequent intervals, in good-sized doses, by the intravenous route.

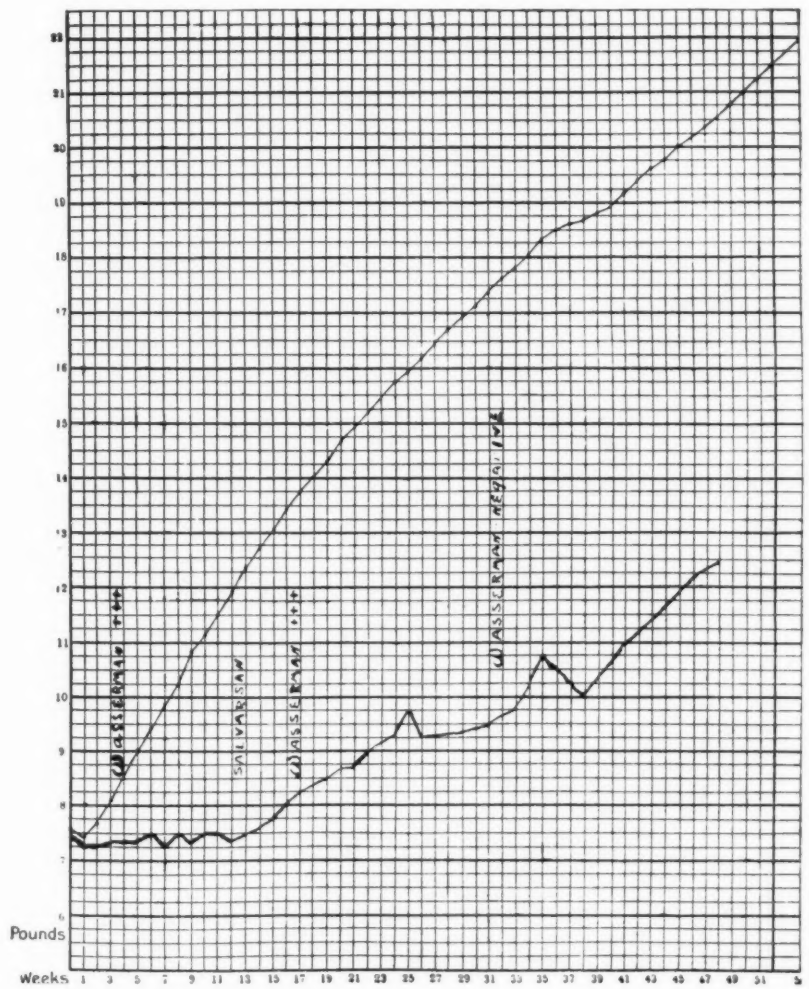

Fig. 6.—Weekly weight chart of H. P., born July 16, 1912. Marasmic type. No clinical evidences of lues. Wassermann negative.

Owing to the fact that so many cases of tardy syphilis show no clinical evidence of the disease in early infancy, we decided to make a study of the marasmic infant so commonly seen in our clinics and hospital wards, with the object of ascertaining, if possible, what percentage

of this group harbored a mild form of lues. We think it possible by the diagnosis and proper treatment, aided by frequent serum tests as the child grows, to ward off some of the later manifestations described above. During the past year every infant coming into the hospital wards and in two of our clinics, showing a stationary weight, with a condition of malnutrition, has had a Wassermann test taken by Dr. Oliver at Lane Hospital. Over one hundred such infants have been under observation, and fifty have been seen every week up to the present time. Eight cases in this group have shown triple + reports, and some others have given a slightly positive reaction. It might be of interest to note here that the cord blood in over one hundred new-born babies has lately been tested by Dr. Oliver, and in a few cases has given a positive Wassermann

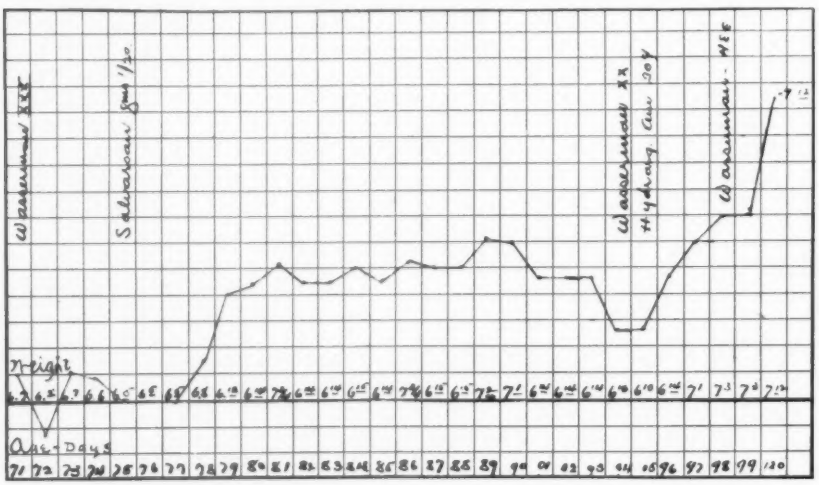

Fig. 7.—L. H. Age on admission 14 days. Birth weight 6.3 pounds. At 75 days, weight 6.5 pounds. Seventy-first day Wassermann +++; 75 day salvarsan; 80 day Wasserman ++; 81 day inunctions hydrarg. ammon.; 120 day, weight 7.12 pounds. Wassermann negative on thirty-first week.

reaction. Several of these cases showed no manifestations of lues either in the mother or child, and he ascribes the positive reaction to some placental effect on the serum.

With the exception that these infants in the marasmic group were poorly nourished and did not show a gain (with a rational formula), none of them had any clinical evidence of lues. As a control, several infants of the same type, but with a negative serum report have been injected also. The ages varied from 6 weeks to 5 months; the formulas remained practically the same before and some time after treatment, and the cases were closely watched by several competent physicians. These cases all received from 1/20 to 1/10 gm. of salvarsan, intraven-

TABLE GIVING DATA CONCERNING TWELVE OF AUTHORS' CASES

Tabulated report of twelve cases of double interstitial keratitis under treatment with salvarsan, showing lack of early symptoms, duration of infection, serum reports after treatment and results of treatment.

Case	Age	F. Hist.	Manifestations: Early: Talked	Walked, Month	1st Tooth, Month	Late: Teeth	Lymph. N.	Ozena	Duration Present Infection, Mo.	No. Recurrence	Salvarsan 1st	Salvarsan 2d	Salvarsan 3d	Serum Reports: Before Tr.	1 Month	2 Months	3 Months	4 Months	5 Months	6 Months	7 Months	8 Months	9 Months	10 Months	Gains in Sight	Cleared	Unimproved
1	6	+	*N	19	13	H	+	..	¾	..	+	+	+	+++	+++	+++	.....	..	....	..	..	—	—	..	+	+	..
2	6	—	N	22	6	H	+	..	..	..	+	..	..	+++	.....	++	.....	..	....	..	..	..	..	..	..	..	+
3	12	+	N	24	..	H	+	..	1	2	+	+	..	+++	.....	.....	.....	—	—	..	—	..	..	..	+	+	..
						M																					
4	5	—	N	..	10	..	..	..	..	..	+	..	..	—	.....	.....	—	..	....	..	..	..	..	..	..	..	+
5	12	—	N	24	7	..	+	..	4	..	+	+	..	—	.....	.....	.....	..	....	..	..	..	..	..	+	..	..
6	12	—	N	14	9	H	..	..	..	..	+	+	..	—	.....	.....	—	..	....	..	..	..	..	..	..	..	+
7	12	—	N	12	8	..	+	+	..	..	+	+	..	—	.....	.....	—	—	—	..	..	..	..	..	..	..	+
8	12	+	N	18	13	..	+	..	¾	3	+	+	..	+++	.....	—	.....	..	....	..	..	..	..	..	+	..	..
9	14	+	N	14	14	H	+	..	12‡	4	+	+	..	+++	+++	—	.....	..	....	..	..	..	..	..	..	..	+
10	4	+	†	36	8	H	+	+	§	..	+	+	+	—	++	.....	—	..	....	..	..	..	..	..	+	..	..
11	16	—	N	24	12	H	+	..	12	5	+	+	..	+++	+++	+++	.....	..	++	..	—	..	..	..	+	..	..
12	7	—	N	..	..	H	+	..	1	..	+	+	..	+++	+++	+++	+++	—	....	..	..	..	..	..	+	..	..

* N = Normal; H = Hutchinson's Teeth; M = Moon molars.
† Talked at 36 months. ‡ Years. § Life.
Case 3 showed an early skin rash.

ously, under mild chloroform-ether anesthesia, and with the exception of slight gastric disturbances in a few cases, no reaction resulted.

In order to simplify matters, we have inserted into this paper four of the eight weight-charts (Figs. 4-7) kept at the clinic. They show the age of the infant at the time of injection, character of Wassermann before and after injection, and the gain in weight.

In the examination of the charts, we find that in each case the child showed a definite gain within six weeks, and usually less, after the injection, with the Wassermann reaction negative after the injection. Two infants having triple + Wassermanns, but without treatment, gained slowly, but six months later had positive reactions.

We believe without question that in many cases of latent syphilis salvarsan produces a beneficial tonic effect. Whether this gain in weight and general condition in infancy is due to this effect or not, we do not know. No doubt the effect is mainly due to the drug on the luetic virus.

As to parallel results with mercurial inunctions, we have not obtained as rapid a gain, nor has the Wassermann reaction become negative as quickly as in our salvarsan cases.

In concluding, we would say that observing these patients for only two years is too short a time to form any definite opinions. It is only by following them up to puberty with blood-tests at intervals, and careful observations as to late luetic manifestations, that conclusions can be made. We believe that many so-called cases of marasmus are due to latent lues, and that if a diagnosis can be made and careful treatment instituted with possibly both salvarsan and mercury, many of the later distressing manifestations of the disease can be averted.

TREATMENT OF RHEUMATIC ENDOCARDITIS

FROM AN OUTPATIENT DEPARTMENT STANDPOINT

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The types of cases of endocarditis which are seen in an out-patient department vary considerably from those which have had simply one attack of rheumatic fever with very slight involvement of the endocardium, to those cases which have had repeated attacks of rheumatic fever with endocarditis. These cases of rheumatic endocarditis may be divided into three classes.

1. Those patients seen in their first attack. These patients have little or no permanent valvular involvement.

2. Those who have had some permanent damage to the endocardium — a type which is often able to compensate for the child's ordinary life; that is, going to school and playing with certain definite limitations to over-exertion.

3. Those who have serious involvement of the endocardium and myocardium. Cases which may be called cripples, for whom there is no hope of complete cure, but who are perhaps compensating enough to allow them to be up and around for short periods of the day, and who are just barely able to come into an out-patient department for an examination or for advice at fairly long stated periods.

4. A class of patients who are so badly crippled that they are unable to go out or to attend an out-patient department safely. These do not enter into our considerations at present, though they offer a family and community problem from another standpoint.

The management of these three types of cases varies considerably.

The patients coming in with their first attack are, from a preventive point of view, by far the most important. These patients demand or should demand far more careful supervision than they get at present. The ultimate seriousness of rheumatic endocarditis must be appreciated when these patients are first seen, and every effort made to prevent their passing into the third class. It is at this time that medical effort and supervision pays most.

We believe that the most important part of the treatment of these patients is prolonged rest. Rest offers the best hope of complete cure if carried out over a long enough period. The hope for permanently curing endocarditis after the first involvement, lessens almost in geometrical progression with each succeeding attack. This rest can be obtained in

two different ways, either in a hospital, or, if the home permits, it can with careful supervision be carried out there.

TREATMENT IN THE HOSPITAL

There are very few hospitals to-day that are alive to the importance of long continued rest for these early cases of rheumatic fever, or that are willing to keep such cases in their hospital beds long enough really to ensure a permanent cure of the endocardium. If not properly treated during the first attack the infection is likely never to be entirely cured. The patient is too often discharged with the infection in a quiescent stage, simply to flare up again under the slightest provocation during a recurrent attack. Hospital treatment to be successful in these cases must be continued over months rather than weeks; the patients being kept in bed and at rest for at least six weeks after all signs of infection, such as temperature, have passed, and then there should be a very gradual increase of activities, the child being under constant supervision throughout the convalescent period. Such treatment can rarely, if ever, be carried out in any of our children's hospitals for acute diseases. We need for this type of case a cardiac sanatorium where the patients can be sent to remain as long as is necessary for a complete cure of their primary rheumatic infection.

The home treatment of such cases will depend almost entirely on the cooperation of the family and the supervision which the hospital offers for follow-up work — a combination which is most difficult. But if this can be carried out satisfactorily, it offers several advantages. From a financial standpoint, it is considerably cheaper than treatment in a cardiac sanatorium. From a standpoint of education it offers the best way of educating the family to the importance of careful supervision in guarding the child against the types of activities which put too much strain on the damaged endocardium or myocardium. We believe that the educational feature of follow-up work in these early cases is most valuable and essential in the after treatment. To a greater or less extent, we must come in contact with the families of these children and educate them to the importance of regulating the lives of these little patients.

Whether the treatment be carried out in the hospital, in the sanatorium or in the home, there are certain points which we think are of considerable value in procuring a successful outcome.

To start with, we must understand the temperament of the child with whom we are dealing. It is almost impossible to prescribe rest or hope to have it successfully carried out unless we realize that with rest we must furnish some means of entertainment. These children should be taught the kind of amusements which they can safely carry on while keeping quiet. This takes careful planning and considerable skill in the person on whom the follow-up work devolves.

In a family in which there are a number of other children, the problem is, of course, more complicated than when there are only one or two other children. In a large city, with a child from the congested district, the question of proper hygienic surroundings, removal from noise and disturbance, from family congestion, are all points which have to be considered, and a working scheme planned out — not one formulated for a short period, but one which is capable of being carried on for months or years.

When these cases can be allowed more leeway then our supervision should not be entirely withdrawn. The medical oversight of the child's life should be carefully gone into with the family. As has been said, family cooperation is absolutely essential, and the *sine qua non* of a successful outcome. The advice of the follow-up worker is necessary to accomplish this successfully. This cooperation should be carried on into the schools by some plan of close cooperation between the hospital, the hospital follow-up workers, the school nurses, the school physicians and the school teachers, so that these cases can be watched for the first sign of any recurrence, and to guard against over-exertion.

The second type of ambulatory cases are those which, after their first or recurrent attacks, have definite organic cardiac lesions for which their hearts are compensating perfectly well. These patients, on physical examination, show a more or less hypertrophied heart, with good clear cardiac sounds, and with good peripheral circulation. The murmur is of less importance than the quality of the heart-sounds. Such patients we treat as ambulatory cases. This type needs definite treatment and supervision from an out-patient department, we believe, along certain lines. Here a complete schedule of living should be mapped out for each child; the amount of activity depending on the capacity of the heart. What the capacity of the heart is, is often a most difficult question to answer, and the only way which we think this can be done is by closely following a child on a given schedule of living, and seeing what the cardiac reaction to this schedule is. Here we need careful physical examination, careful medical instruction and a systematic and complete follow-up program, our follow-up worker keeping in close touch with the family as well as with the child. Here again the education of the family, gaining their cooperation and intelligent understanding of the child's condition, is absolutely essential to the successful management of such a case. Here also there must be close cooperation between the hospital, follow-up worker, school nurse, school physician and teachers, so that the child's condition is understood and watched from all sides. There is very little use in the physician in a hospital mapping out such a schedule of life without going to the trouble of having cooperation in every sphere which the child touches — home, recreation, school, rest, eating and sleeping. The educational and vocational sides of these children are just as impor-

tant as the home routine life, and unless we can enter the recreative side of the child's life and furnish the type of recreation which the child will enjoy, and which at the same time is open to the child to enter into, we cannot hope, from a psychological standpoint, to have its assistance. For, after all, we are dealing with human nature as well as with an organic condition, and to benefit the one we must meet the other and supply its definite and legitimate needs; so that we feel that the problem of furnishing the proper type of recreation is a most important one, and one which is almost entirely neglected in the schedules outlined for such cases, or at least our means for putting it into action are not fully developed.

Most of this devolves on the follow-up worker, and it is on her ability to work out the kind of recreation which meets the particular child's needs, and then supply this in some definite and tangible form, that success will depend. She must gain the hearty cooperation and understanding of the school authorities. Very often we order such children to stay out of school, although we believe that if their school activities and demands were limited, and not as much were expected of these children by the school authorities, that we would do better to send them to school than to keep them at home, and have the continuous over-sight of them at home, which is almost impossible and very often impracticable in the conditions under which they live.

When these children suffer from relapses, our treatment must again in these cases be either rigid hospital care, cardiac sanatorium care, or home treatment in bed. Here again the period of rest must be long, and the supervision most carefully worked out. Every time these cases have a recurrence, their chance of ultimate recovery is that much lessened, so that we must hope far more from our preventive than from our curative methods. Damage once done can never be entirely compensated for, and gradually these cases, unless most carefully handled, drift into the third type of cardiac cripples, which are almost the most pitiful class of children met with in a medical children's hospital. For such children there is practically no hope of cure; their lives, however, can be made more comfortable and useful in a great many instances if they can be properly controlled and a scheme of life mapped out. Here again, the most important feature is continued rest, staying within the powers of the cardiac muscle. Over-straining, over-activity, over-doing, mentally or physically, are the most important things to be guarded against. Such cases should be in constant touch with the hospital follow-up worker, going into their homes, planning their amusements and mapping out some course of study or vocational work by which these cases can be kept contented and their lives made useful. Many such cases can be taught not only to enjoy their existence, but to be sources of income to their

families, and so avoid the feeling that they are drains on their family, which is a most important point for their mental serenity.

The medical supervision of these patients is important in following the cardiac condition and making the necessary changes in the schedule of life, carefully watching that we are not over-taxing the myocardium, and yet giving the children as much exercise as it is possible for them to have.

In all of these cases medical treatment plays a minor part as compared with rest. As in tuberculosis fresh air plays the most important part in a successful treatment, so in these cardiac conditions rest is the key-note for successful treatment. We must have fresh air and good food combined with rest, as well as a calm, cheerful mind.

MEDICAL TREATMENT

The medicinal treatment is symptomatic. In the primary attacks, cardiac stimulants are practically never needed. Salicylates, usually in the form of acetyl-salicylic acid (aspirin) with sodium bicarbonate, are of considerable value during the acute febrile stage, and the use of salicylates from time to time may have a preventive function, especially when the patients are exposed to any infection or have been exposed to sudden changes of weather. The removal of any infection should be carefully looked into. The wholesale removal of tonsils and adenoids, we do not think is necessary, but we feel that the removal of diseased tonsils and adenoids is essential; further, that the care of teeth, removal or attention to carious teeth is equally essential, since these are just as important a source of infection as are diseased tonsils in many cases. Dental care, we feel, should receive more attention in the treatment of such cases. The diet, with special attention to constipation, should be carefully supervised. Carefully prepared diet lists and the keeping by these children of diet books putting down for certain days in the week just what they have eaten, we have found to be of considerable value in the way not only of being able to change their diet to a more rational one, but also of giving one more avenue for home education, which is one of the most important points in handling these types of cases. They should also keep a record of the periods of rest and what type of exercise they have taken each day, which will help to give an accurate estimate of their daily lives. We find that this is very much more practical than simply telling the children or explaining to their mothers what we wish done. All our directions are written out. The schedule of life is given to them in written form; that is, they are supposed to rest so many hours, stating the different periods, and what time of day these periods should come, describing what we mean by rest, the types of activities permitted and what are not permitted, and then having them keep a record of just what they do, and how long they rest. We find this a very practical

method of keeping in touch with these cases, and it arouses their interest and gains their cooperation when the more simple method of verbal direction would surely fail.

In cases which present themselves with well developed cardiac lesions, but with, as yet, no failure of compensation, the same observation obtains and the daily life is so regulated that the least strain possible is put on the heart. The diet is regulated; thorough mastication is urged, the bowels kept moving regularly. The patient is advised to report on the first signs of fatigue, pain, loss of appetite or dyspnea.

When a patient comes to us with broken compensation the *sine qua non* is rest, and with out-patients we have found it necessary to exaggerate the severity of the case and consequently the necessity of resting more than is really essential. It is difficult to make this class of patients and their families realize the seriousness of heart lesions, and many of these children have to be brought to the hospital in order that we may be sure the most rigid care is being observed.

The patients who are able to be up and going to school, but who are beginning to show slight signs of failing compensation, as a routine are given a half hour's rest between morning school session and lunch, and one hour after returning from school in the afternoon. Bed at 7 or 7:30 p. m., combined with digitalis drops 2 to 5 after meals. If this be not sufficient the child is taken from school and given two hours in bed, morning and evening, and the dose of digitalis raised until control is obtained.

During the past year and a half we have been having a special cardiac clinic in the Boston Dispensary. The extent of the clinic is shown in the following table:

Diagnosis.	Cases
Endocarditis	148
Pericarditis	1
Myocarditis	13
Chorea (with cardiac involvement)	101
Chorea with endocarditis	41
Aortic stenosis	1
Aortic regurgitation	8
Mitral regurgitation	59
Chronic endocarditis	31
Acute endocarditis	6
Mitral stenosis	2
Mitral regurgitation and stenosis	23
Congenital	8
Functional	10

One of us is in daily charge of this clinic and is in touch with all the work through our social service worker. In this way we are in touch with the family problems, plan the recreation and vocational and school life of the child as far as it is possible. All the agencies that are interested in these children are drawn into the program of life mapped out for each child.

It was not until we took up the study of these cases that we became alive to the large number of children who fall into this class, and the extent of our problem has been growing ever since. We feel that similar attempts to study the cardiac problem should be undertaken in other cities and in all children's hospitals. The plan which we would suggest for a study of cardiac cases among children would be as follows:

There should be first of all, a general survey of the actual number of cardiac patients attending the hospitals. If made a city investigation, of course, this would include the records of various hospitals and of the public schools.

Second, an estimate of the number of children with the first attack who should be in some institution, as a cardiac sanatorium, for a shorter or a longer time. How large this number is it would be impossible to state, of course, without such an investigation, but that such a sanatorium is needed in every community is beyond any question of doubt.

This sanatorium should be built on the cottage plan, because we do not think it would be advisable to have a large institutional building for such cases, but that they would do better if they were grouped by ages and more or less according to the temperament as well as according to the extent of the cardiac lesion and the amount of activity allowed. Some cottages would be, in this way, for cases that could take very little exercise, other cottages for those who had graded up to a considerable amount of exercise and were just about ready to be discharged.

Third, an estimation of how many of these cardiac cases could be cared for at home, if they were properly supervised and followed up as outlined above.

Fourth, an estimate of the number of practically incurable cases and cardiac cripples should be made, and some means of reaching these children educationally, vocationally and recreatively should be attempted. For they can have a great deal done for them to make life comfortable, and in many cases even by a proper kind of training along certain vocational lines, these patients can be made self-supporting, and their lives made comfortable to themselves and no longer a financial burden to their families.

In order to study this problem in a broad way, there must be the cooperation of certain groups of workers — the hospital physicians to give their medical examinations; the school physicians to give theirs; the social workers and nurses to supervise the plan outlined by the medical agency in charge of the cases; the cooperation of the school board, hospital trustees, hospital managements, convalescent homes, family and community in supporting such work.

NEUROTIC VOMITING IN AN INFANT*

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Persistent vomiting in infancy is usually attributable to one of two general causes, viz.: 1. Inability of the stomach to digest the food. 2. Malformation of, or partial obstruction in the esophagus or stomach.

In the first-named condition there is obtainable in practically every case, a history of incorrect feeding. The most common errors are:

1. Improper food.
   a. High fat.
   b. High sugar.
   c. Solid food at a too early period.
2. Too frequent feeding.
3. Too much food.

Vomiting is usually not the only symptom in these cases of gastric indigestion. Intestinal disturbances, as shown by presence of constipation or diarrhea, follow. The appetite is usually poor, but the reverse may be true. The vomiting may occur at any time after feeding and is frequently accompanied by repeated regurgitations of small amounts of food and eructations. The vomitus is usually sour and contains an excessive amount of mucus. Cure follows only when proper feeding is instituted.

Marked malformation of the esophagus or stomach with complete obstruction of the canal is obviously incompatible with life. When, however, the stenosis is not too great, the child may live over a long period and vomiting is then the chief subjective symptom.

In esophageal obstruction, regurgitation usually occurs soon after swallowing. Solid food reaches the stomach with more difficulty than does liquid food and in some cases cannot be taken at all. If a diverticulum is present, food may be returned which had been taken several days previously.

Diagnosis is often aided by the finding of an obstruction on attempt to pass the stomach tube, or by Roentgen ray after bismuth feeding.

The following malformations of the stomach may cause persistent vomiting:

1. Stenosis of the cardia.
2. Hypertrophic stenosis of the pylorus.

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* From the Harriet Lane Home for Invalid Children of the Johns Hopkins Hospital.

* Submitted for publication Oct. 23, 1913.

3. Hour-glass stomach. (Bilocular stomach.)

In stenosis of the cardia the symptoms resemble those of esophageal obstruction.

Hypertrophic stenosis of the pylorus, by far the most common cause of obstruction in the stomach, would seem to be of two types: one in which the change is chiefly organic, the other in which spasm plays the greater rôle.

The onset of vomiting in hypertrophic stenosis of the pylorus is ordinarily during the first few weeks of life; rarely later than the sixth week. The vomiting is very forcible, occurs usually soon after the feeding and is seldom repeated until the food is again taken.

Peristaltic waves are seen passing slowly from left to right across the epigastrium in nearly every case. These waves are usually best seen soon after taking of food and before vomiting occurs. Constipation and small stools are the rule. There is no fever. A tumor is palpable in the region of the pylorus in cases in which marked hypertrophy is present.

With an hour-glass stomach, when the stenosis is marked, the symptoms approach closely those of pyloric stenosis.

There is another more or less well recognized type of persistent vomiting which has been described by various writers under the name of habitual vomiting, nervous vomiting, neurotic vomiting, etc.

This type is characterized by the presence of vomiting after each feeding without any other symptoms of gastric of intestinal disturbance. The vomiting is not forcible. There are generally present other evidences of a neurotic disposition. Changes in the proportion of fat, sugar and protein in the food have no effect on the vomiting.

This vomiting is usually considered of little importance, and not likely to lead to serious harm to the child. In the case here reported the duration of the vomiting, the critical condition to which it brought the child and the prompt response to treatment were especially noteworthy.

CASE REPORT

History.—The patient, Doris H., born April 29, 1912, was admitted to the hospital Nov. 18, 1912.

The family history was unimportant. The mother and father were well. There was one other child aged 2½ years, who had always enjoyed good health.

The patient was normal at birth and weighed 8½ pounds. The baby was breast-fed for 3½ months. She was nursed every two hours until after the second month, and then the night feedings were reduced to two or three. The infant thrived while getting breast-milk and at the end of this period is said to have weighed about 13 pounds. There was no vomiting and the stools were normal.

The baby was then weaned from the breast by gradually substituting a malted milk formula (2 teaspoonfuls to 3 ounces of boiled water), so that by the end of ten days the breast-milk feedings had been wholly replaced. Vomiting began with the change to artificial food. During the entire period of weaning the breast milk was retained while part of every artificial feeding was vomited.

Mellin's food, Nestlé's food, condensed milk, modified cow's milk, and finally breast-milk secured from a wet nurse were successively tried, but the vomiting was constantly repeated after each feeding.

For some time previous to admission to this hospital the following formula had been given:

Whole milk	10½	ounces
Boiled water	22	ounces
Milk sugar	1	ounce

Divided into ten feedings, given at two-hour intervals, two night feedings being omitted. This mixture was also vomited in the same manner as were the other foods.

The vomiting is said to have occurred usually twice after each feeding and without any evidences of discomfort. The vomitus consisted ordinarily of the food in the form given. It was never green, only occasionally in curds and of a sour odor. Since the onset of the vomiting there had been a slow, but continuous loss of weight. There had been no distention of the abdomen, no fever, and the child had always slept well.

Examination.—On examination in the ward the patient was found to be an exceedingly poorly nourished and somewhat underdeveloped child. Weight 7 pounds, 13½ ounces. Height 23 inches. Temperature 95.5 F. The muscles were flabby and the skin was relaxed, inelastic and hung in folds. The anterior fontanelle was depressed. The eyes, ears and nose were normal. The tongue was clean. There were no teeth. The pharynx and tonsils showed nothing abnormal. The lungs were clear. Von Pirquet test negative. The heart sounds were weak but regular. There was no cardiac murmur.

The abdominal wall was thin, but the muscles were of fair tone. The spleen and liver were not enlarged. There was no tumor in the region of the pylorus and no peristaltic waves were visible. The extremities and reflexes were normal.

Management.—On admission 4 ounces of the following mixture was given: Whole milk, 10 ounces; barley water, q. s., 24 ounces. This was well taken, but practically all vomited within a period of twenty minutes. Then 4 ounces of barley water was given at four-hour intervals. Two feedings of this were taken and retained, but the third was refused.

Next, skimmed milk (1 part) with barley water (2 parts), 4 ounces every four hours, was given and vomiting followed each feeding.

The quantity was then reduced to 2 ounces, but still the vomiting continued. Feedings given by gavage were retained no better, even when this was preceded by lavage.

The baby took the food greedily and rapidly when permitted. The region of the stomach became prominent and the normal gastric tympany became dulled. After a few minutes a sucking motion of the lips and a chewing motion of the jaw began. Then in a short time, usually fifteen minutes to one-half hour after the feeding, a part or all of the food was slowly returned, and when the mouth was filled, flowed slowly out over the cheeks. The motion of the lips and jaw continued during the vomiting. Sometimes the vomiting was repeated.

Occasionally the child would reswallow a small portion of the food, but this was not a constant, or a usual feature. The vomiting was at no time forcible; in fact, was more in the nature of regurgitation. At no time was there any evidences of pain or nausea. No peristaltic waves were seen on frequent and careful observation during and after feedings. There was no mass felt in the region of the pylorus. The food was taken eagerly and the child always seemed hungry, even immediately after vomiting.

On November 23, five days after admission, the child weighed 7 pounds, 10½ ounces. Her general condition seemed worse. She was much quieter and the abdominal muscles more relaxed. The temperature was subnormal and pulse 96 to 118 per minute.

At this point a semisolid food was given with the idea that the child would be unable to regurgitate it in the manner she had the liquid food. For this purpose a thick barley gruel was given with a spoon on November 23 and it was retained. The next feeding was also retained, but a portion of the third feeding was vomited. The other feedings of that day were retained. On November 24 milk was added to the gruel and at once this liquid food was vomited. On November 25 a very thick porridge, prepared from one part of farina and 6 parts of whole milk, boiled together for one hour, was given. The resulting food, when cool was quite stiff; so much so that it would adhere to an inverted spoon. Two ounces of this porridge was given by a spoon every four hours at first and after a few days the amount was increased to three ounces.

The baby took this food well and there was an immediate decrease in the frequency of vomiting and of the amount vomited. Vomiting became the exception and soon ceased entirely. Sterile water, given at the beginning of the feeding of farina, was at times vomited, but later this also was retained.

With the giving of this solid food there was a rapid and steady gain of weight, except for a few days (December 16 to 18) following a febrile attack (acute rhinopharyngitis, probably influenzal) at which time there was a return of

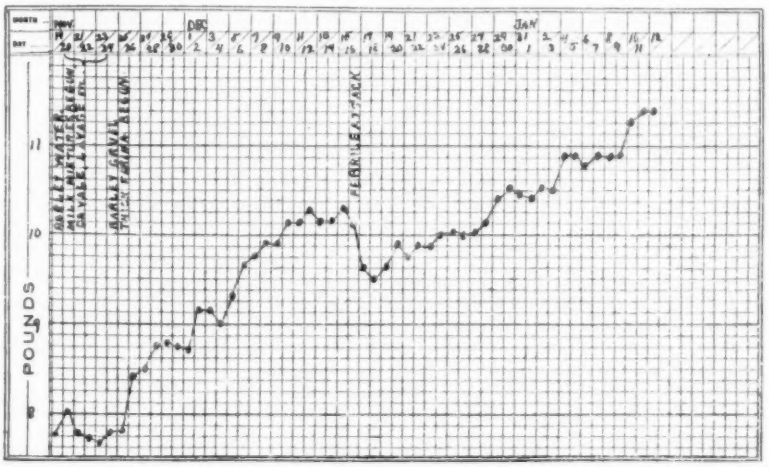

Chart showing weight-curve in Doris H., aged 6 months, with neurotic vomiting.

vomiting and a rather sudden and marked loss of weight (12 ounces being lost in three days). The stools remained normal during the farina feeding and tests for starch in them (iodin method and microscopic examination) were negative.

The child left the hospital Jan. 12, 1913, at which time she weighed 11 pounds, 6 ounces, a gain of 3 pounds, 11½ ounces from the time of beginning the feeding of the solid food, Nov. 23, 1912.

During the stay in the hospital the child developed a head-rolling habit which occurred with much frequency when awake, especially if there was any stir about the ward, as at feeding times, ward rounds, etc.

Subsequent Course.—The patient was seen April 29, 1913, three and one-half months after discharge from the hospital. At that time she weighed 15 pounds, 3 ounces. There had been no return of the vomiting and the stools had remained normal. Farina had been the only food given since leaving the hospital.

April 29 orange juice, scraped beef, oatmeal, zwieback and soft boiled eggs were added to the farina diet. A report from the mother dated Aug. 7, 1913, stated that the child had vomited on a few occasions only, immediately following

additions to the diet. She was doing well and weighed 19½ pounds. The bowels were regular and stools normal.

DISCUSSION

The essential features of the case, briefly stated, were:

1. The sudden onset of vomiting at the age of 3½ months, with retention of breast-milk while artificial food was vomited.

2. The persistence of the vomiting despite the great variety of liquid foods given, with cure resulting promptly on giving semi-solid food.

3. The slow, forceless and apparently willful nature of the vomiting without reswallowing.

4. The neurotic disposition of the child, as evidenced by the development of a head-rolling habit.

In seeking an explanation for the vomiting as observed in the case related above, the following conditions were considered and excluded:

1. Chronic gastric indigestion.

2. Hypertrophic pyloric stenosis.

3. Malformation of the esophagus.

4. Rumination.

Chronic gastric indigestion was discarded because of the utter lack of any other evidence of alimentary disorder. The vomitus was usually unchanged food; the stools were normal; the tongue was clean and the appetite good. Sudden and complete cessation of vomiting and rapid gain of weight followed the giving of semi-solid food.

Hypertrophic pyloric stenosis was excluded on the grounds of the sudden onset of vomiting at 3½ months *with*, at the beginning, *vomiting* of artificial food and absence of vomiting of breast-milk. Projectile vomiting, peristaltic waves and a palpable tumor were also absent.

Malformation of the esophagus, either congenital or acquired, could well be excluded. The child retained breast-milk at first, while vomiting other liquid food. There was no difficulty in swallowing a very thick food, and no obstruction encountered in passing the stomach tube.

Rumination was excluded in this case because of the absence of the characteristic reswallowing of the food.

The usual causes of persistent vomiting having been satisfactorily excluded, we were led to explain the vomiting as a neurotic manifestation. The apparent willfulness of the vomiting, as suggested by the movements of the lips and jaws, with the seeming satisfaction at its completion; the mode of cure (the baby, for a long time after feedings of farina were given, continued the movements of the lips and jaws, but was unable to regurgitate this semi-solid food); and the evident neurotic constitution of the child, as shown by the acquiring of the head-rolling habit, supported this explanation.

The type of vomiting in the cases of rumination described by Aschenheim,[1] and Lust,[2] was much like that in this case, but as has been stated before, here the characteristic reswallowing of the food was absent. Hahn[3] has described several cases of habitual vomiting which he cured by giving a porridge of milk with about 5 or 6 per cent. of ground grits.

It seems not unlikely that there is a considerable number of instances of chronic vomiting of this type which either go unrecognized or, when recognized, are found difficult to cure. This form of treatment could probably be used successfully in a large percentage of similar cases. That a solid food of the nature given can be taken and utilized by an infant of 6½ months is well illustrated by this case. The child thrived on this food over a period of more than five months (gained 7 pounds, 6½ ounces). There was no digestive disturbance and no starch demonstrable in the stools.

I wish to express my thanks to Dr. John Howland for the privilege of reporting this case.

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2. Lust: Zur Pathogenee der Rumination im Säuglingsalter, Monatschr. f. Kinderh., 1911, x, No. 6; Orig. ii.

3. Hahn, Hans: Zur Therapie des habituellen Erbrechens der Säuglinge, Med. Klin., 1911, vii, 1452.

PROGRESS IN PEDIATRICS

RÉSUMÉ OF WORK ON TUBERCULOSIS IN CHILDREN FOR 1913 *

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MODES OF INFECTIONS

1. Medin, O.: The Modes of Infection of Tuberculosis in Infants, Arch. f. Kinderh., 1913, lx, lxi, 482.

2. Calmette, A.: The Modes of Infection of Tuberculosis, Rev. de la tuber., 1913, No. 5, p. 321.

3. Emrys-Roberts, E.: The Portals of Entry of the Tubercle Bacilli, Brit. Med. Jour., 1913, i, No. 2718, p. 210.

4. Findlay, L.: The Portals of Entry of the Tubercle Bacilli, Ztschr. f. Kinderh., 1913, vii, 5-6, p. 503.

5. Findlay, L.: The Intestine as the Pathway of Infection of the Tubercle Bacillus, Brit. Jour. Child. Dis., 1913, x, 119, p. 502.

6. Ritter, J.: The Pathogenesis of the Glandular or Secondary Form of Infantile Tuberculosis, Illinois Med. Jour., 1913, xxiii, 58.

7. Konschegg, A. V., and Lederer, R.: Contribution to the Clinic and Pathology of Pulmonary Tuberculosis in Infants, Monatschr. f. Kinderh., 1913, xii, 3, p. 140.

8. Trautmann, G.: Tuberculous Cervical Lymph-Nodes Infected from the Tonsil, München. med. Wchnschr., 1913, lx, 16, p. 866.

9. Thomson, G.: Teeth in Relation to Pulmonary Tuberculosis, Med. Press and Circular, 1913, xcv, 38, 44, p. 33.

10. Holt, L. Emmett: Tuberculosis by Direct Wound Inoculation, Pediatrics, 1913, xxv, 5, p. 315.

11. Dufour, H., and Thiers, J.: Transmission of Tuberculosis from Mother to Fetus, Ann. d. méd. et chir. inf., 1913, xvii, 13, p. 435.

12. Harbitz, F.: Congenital Tuberculosis, München. med. Wchnschr., 1913, lx, 14, p. 741.

13. Zarfl, M.: Congenital Tuberculosis, Jahrb. f. Kinderh., 1913, lxxvii, 1, p. 95.

O. Medin[1] studied the autopsy reports of 7,630 children who died in their first year at the Stockholm Hospital from 1842 to 1911. His aim was to throw some light on the mode of tubercular infection. Six hundred and twenty-three of the children had tuberculous lesions, but this does not mean that they died of the disease. In only six of the 623 cases were the lesions found exclusively in the intestines or mesenteric glands; 313 had combined lesions, but those farther advanced were in the bronchial glands and lungs.

Medin discusses the subject under the following heads: (1) Conceptional, or intra-uterine infection. Of the 623 cases only one was suspicious of being of congenital origin. (2) Infection through the skin. This is very rare in young children, and when it occurs is usually

* Submitted for publication Dec. 12, 1913.

secondary to a tuberculosis of the internal organs. (3) Infection through the mucous membranes: There was no instance of this mode of infection, and the writer concludes that the mucous membrane of nose and pharynx play a very insignificant rôle in the production of tuberculosis. (4) Infection through the larynx: This occurred in one case. (5) Primary pulmonary tuberculosis: In practically 98 per cent. of the cases the infection was primary in the lungs and occurred by inspiration of the infecting material. (6) Primary intestinal tuberculosis: In only 2 per cent. were the intestines the primary seat of the lesion and the rarity of this mode of infection in very young children may be inferred.

Medin also studied the histories with reference to the milk supply, but found that all the cases occurred in one section of the hospital which was supplied throughout with milk from the same source. In his thirty years of experience as pediatrician he has never seen a case of tuberculosis which could be ascribed to infection through the milk.

A. Calmette[2] again publishes his views on the mode of tubercular infection. Direct hereditary transmission from the mother he finds possible, but very rare, and it has never been proved that the father can directly transmit the infection. Infection through the mucous membranes can take place at any age. The mucous membrane of the eye allows easy passage to the tubercle bacilli. The migrating leukocytes engulf the bacilli and then re-enter the blood or lymph-stream, carrying them to various parts of the body. The lung can become directly infected by inhalation of air impregnated with bacilli. Calmette says it is impossible to establish a difference between a primary aerogenous infection and an infection brought to the lung by the lymphatics.

The digestive tract furnishes an easily accessible atrium of infection. The bacilli travel the entire length of the tract without producing any lesion; they are taken into the lymph-channels and hence into the general circulation. The number and virulence of the infecting bacilli determine whether the resulting lesions are single and latent or multiple and grave. The lungs are usually the seat of the latter because of the anatomic relations of these organs.

E. Emrys-Roberts[3] agrees with Calmette that the most frequent portals of entry of the tubercle bacilli are the mucous membranes of the digestive and respiratory tracts, but he cannot agree that of these the digestive tract is the most frequent. As to ante natal infection, he feels that there is a strong probability of it. He says that as pregnancy with its stress and debilitating effects render the organism less able to cope with infection, it would not be surprising, if in the circulating blood of the mother were found tubercle bacilli in far greater numbers than under normal conditions. Moreover, as the cells of the intestinal mucous membrane cannot prevent the passage of the tubercle bacilli,

it is difficult to see how the cells of the chorionic villi can prevent their passage into the fetal circulation. The author says it is easily surmised that the bacilli introduced into the fetus may be relatively few in number; that the lesions they produce may be perifollicular and retrogressive; that the v. Pirquet test may be negative; and while all this makes the diagnosis of antenatal tuberculosis difficult, yet its possibility cannot be denied.

L. Findlay[4] performed experiments to ascertain the portal of entrance of the tubercle bacillus, and the relative infectiousness of the human type for the respiratory and digestive tracts. He easily produced pulmonary tuberculosis in rabbits by causing them to inhale air impregnated with the tubercle bacilli. Feeding experiments, on the other hand, were not so successful because of the danger of the bacilli entering other organs at the same time. In feeding through a sound there is always danger of insufflation and by opening the abdomen the peritoneum is easily infected. The author intends in the future to enclose the bacilli in gelatin capsules and pass these through a tube into the stomach. He does not feel that his experiments confirm the statement that tuberculous infection often occurs by passage of the bacilli through the uninjured intestinal mucosa.

L. Findlay[5] performed a second series of experiments to ascertain what part the intestines play as a portal of entry of the tubercle bacillus. Cultures of bovine and human bacilli were suspended in oil, enclosed in gelatin capsules and injected through a stomach tube into the stomach of healthy rabbits and rabbits which had recently suffered from intestinal catarrh.

Findlay draws the following conclusions from his experiments:

1. Healthy rabbits can be infected by ingestion of large amounts of bovine tubercle bacilli.

2. The bacilli can pass through the apparently intact intestinal mucous membrane and reach the mesenteric glands within a period of six days.

3. When infection occurs the intestine is invariably the seat of lesions, and thus tuberculosis of any organ, other than the intestine, is always a secondary infection when the bacilli have entered by the intestinal route.

4. Catarrh of the intestine does not favor the passage of the tubercle bacilli through the wall, but allows of a more constant and wide-spread infection and in this way facilitates dissemination.

5. Healthy rabbits apparently cannot be infected by this ingestion of large amounts of the human tubercle bacilli.

6. Rabbits just recovered from intestinal catarrh develop tuberculosis after the ingestion of human tubercle bacilli.

7. With the human type of organism a local lesion, though always present, may be slight in comparison with the diseased foci in the mesenteric glands.

J. Ritter,[6] discussing the pathogenesis of glandular tuberculosis in children, says that modern clinicians and teachers hold to the theory that primary tuberculosis infection with few exceptions occurs during the first months of the child's existence, or during the fifth or possibly the sixth year; that the primary focus, generally aerogenous in origin, is in the lung itself near a bronchus but usually subpleural; that after a shorter or longer period of incubation the regional glands are primarily involved and the cervical glands secondarily; that subsequent forms of tuberculosis, pulmonary, osteal, and meningeal, are tertiary manifestations of the disease.

Only occasionally is the infant infected before birth; usually he is slowly and gradually infected from living in a tuberculous environment during the first years of life. If the number of bacilli taken into the organism is small and the infection only occasionally repeated, a cure or arrest of the tuberculous process will take place and there will be produced a gradual immunity which will protect against phthisis in later life. If, however, many virulent bacilli are taken into the organism and the infection is often repeated, the child will succumb to a generalized infection. The immunity acquired by infection with small numbers of bacilli is, however, only relative, for, if the organism becomes reinfected with a large number of virulent bacilli, a so-called massive infection, there is established an active tuberculous process which usually terminates in death.

A. V. Konschegg and R. Lederer[7] report the pathological findings in five cases of pulmonary tuberculosis in infants. In four there was a pulmonary caseated lesion and tuberculous changes in the bronchial lymph-nodes. The changes in the glands were of more recent date than those in the lungs. These findings substantiate those of Küsz, Albrecht, and Ghon, that the primary lesion is in the lungs and the secondary in the lymph-nodes.

The changes in the lungs in the fifth case resemble those of a chronic phthisis in the adult.

G. Trautmann[8] is convinced that cervical lymph-nodes are frequently infected through the tonsils. In this condition, therefore, not only the affected glands, but the tonsil as well should be removed. It must be remembered in this connection that the tonsils sometimes appear quite normal, yet after extirpation are found to be the seat of tuberculous lesions.

G. Thomson[9] maintains that the prevention of dental caries will do more directly and indirectly toward decreasing tuberculosis than any

other measure that has yet been conceived. In elaborating this theme he discusses the development and care of healthy teeth and the means of preventing dental caries.

L. Emmett Holt[10] says that, though wound inoculation is not a frequent method of contracting tuberculosis, it occurs often enough to be a real danger. The case he reports is of more than ordinary interest because of the virulence of the infection. The infant, aged 3 months, was admitted to the hospital with extensive ulceration of the penis. When he was 8 days old, ritual circumcision had been performed and the blood sucked in the usual way. The wound did not heal properly and at the end of a week suppurated. Ulceration then began in the prepuce and extended steadily. Two weeks after operation glandular swellings were observed in the groin. For two weeks before entrance to the hospital there had been a steady loss of weight, anorexia, slight fever and cough. The last few days before entrance a maculo-vesicular rash had developed over the trunk and extremities. Examination showed a well-nourished child weighing 11½ pounds; over the body were scattered about twenty lesions resembling varicella. Many râles were heard over both lungs; the inguinal glands were large and nodular; the entire penis was destroyed by an ulcerative process. The tuberculin skin reaction was positive and tubercle bacilli were found in the discharge from the penis, the sputum and in the scrapings from the cutaneous lesions.

The child lived sixteen days. At autopsy practically every organ was found to be the seat of tuberculous lesions. Scattered throughout the lungs were miliary tubercles, as well as over the base of the brain. The spleen, liver, kidneys and adrenals contained tuberculous nodules. The small intestine was ulcerated, the bronchial, mediastinal, inguinal retroperitoneal glands were caseated. Of especial interest were the changes in the heart. Small yellow tubercles were found on the coronary arteries and tuberculous nodules in the walls of the right ventricle — lesions never before observed by the writer. Cheesy tubercular nodules in the adventitia of the iliac artery were also interesting and unusual findings.

H. Dufour and J. Thiers[11] report the following, in support of the theory of transmission of tuberculosis from mother to fetus:

A woman, 19 years old, was admitted to the hospital with advanced tuberculosis of both apices. She died shortly afterward with symptoms of meningitis. The autopsy showed besides a tuberculous meningitis and tuberculosis of the left lung (cavities at the apex and miliary tuberculosis throughout the lung) an impregnated uterus. The abdomen of this fetus was large and distended with fluid. The placenta showed macroscopically and microscopically no tuberculous lesions.

The fluid from the abdomen of the fetus was injected into guinea-pigs, as was also a fluid obtained by titrating the placenta in normal salt solution. The first guinea-pig was killed after twenty-three days and the spleen showed three small tubercles containing many tubercle bacilli. The other guinea-pig died four days after inoculation from peritonitis. The serosa was congested and the intestinal coils were agglutinated. There was a grayish exudate at the point of inoculation.

F. Harbitz[12] reports this case of congenital tuberculosis. Twenty-eight days after the child's birth the mother died of tuberculosis. There was a tuberculosis of the endometrium at the placental site, so that transmission of the tubercle bacilli directly into the blood-stream could easily have occurred. The uterus had probably become infected from old tuberculous lesions found in the tubes.

The child was removed from the mother directly after birth, but died on the twenty-fifth day. Extensive tuberculous changes were found in the lungs and lymph-nodes.

Harbitz would gather from daily experience that an infant dies very shortly after contracting a tuberculous infection, and, if a child of one to three months has lived even for a short time in a tuberculous environment, no conclusions can be drawn as to whether the infection occurred before or after birth. On the other hand, infants with congenital tuberculosis can live a quarter to one year or even longer.

Harbitz examined at autopsy six infants less than five weeks old, all born of tuberculous mothers, and found in none evidence of tuberculosis. Careful autopsy examinations of a great number of cases of tuberculosis gave evidence of acquired disease and spoke in favor of the view that infection before birth is extremely rare.

M. Zarfl[13] reports a positive von Pirquet reaction in a seventeen-day-old child, the earliest ever obtained. The mother of the child was tuberculous. The child's weight decreased, the liver and spleen and inguinal lymph-nodes became enlarged, but the lungs remained negative. The early appearance of the positive von Pirquet was proof that the disease was congenital, for after infection at least four weeks must pass before a positive tuberculin reaction can be obtained.

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21. Nobécourt and Darre: Tuberculous Bacillaemia in Children, Presse méd., 1913, 3, p. 26.

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J. Fraser[14] studied the type of organism in a series of seventy cases of bone and joint tuberculosis in children. The tuberculous tissue obtained at operation was inoculated subcutaneously into animals. At the end of six weeks the animals were killed and extensive tuberculous changes were found in the glands and internal organs. The organism was grown from these lesions. The diagnosis of the organism was based on five tests. (1) Cultural characteristics; (2) morphological appearances; (3) growth on glycerin-egg media; (4) reaction to glycerin bouillon; (5) results of rabbit inoculation.

In forty-one instances the bovine bacillus and in twenty-six the human bacillus was found. In three both types occurred.

In twenty-three of the cases in which the bovine bacillus occurred the material came from children below three years of age who had been fed almost entirely on cow's milk. Fraser says there can be no doubt of the milk being the medium of infection.

In fifteen of the cases from which the human bacillus was isolated there was a definite family history of tuberculosis.

Fraser draws the following conclusions: (1) It is possible by certain tests to distinguish between human and bovine types of tubercle bacilli. (2) Considerable proportion of bone and joint tuberculosis among the children of Edinburgh is due to infection with the bovine bacillus. (3) The bovine bacillus is introduced into the body by the drinking of infected milk. (4) A large proportion of the cases due to human bacilli are the result of direct infection by a consumptive co-resident.

A. de Besche[15] inoculated guinea-pigs with macerated cervical and mesenteric glands from 134 children who had died in the Cristiana Hospital. In fifty-two of the children a tuberculous process was found at autopsy. In twenty-eight it was the cause of death; in fourteen a secondary finding, and in ten a latent process. Forty-six of the guinea-pigs reacted to the injection and in most instances those injected with material from the children who died of tuberculosis. Moreover, the guinea-pigs reacted to injections of both cervical and mesenteric glands, and Ungermann's statement, that usually, when there is a tuberculous infection of the lymph-nodes the process is a wide-spread one, is confirmed. As more positive reactions occurred with material from older children, the increase in the frequency of tuberculosis with age was shown.

The type of organism was studied in the fifty-two cases in which the tuberculous process was the cause of death. In forty-five it was the human type; in three, the bovine type; in one the culture showed both types, and in three the type could not be determined.

J. Orth[16] believes that the two types of tubercle bacilli, bovine and human, cannot be sharply differentiated; that transitional types exist, and transformation from the bovine into the human type can take place. He says that tubercle bacilli of the bovine character occur rarely in the adult, but are found on an average of 10 per cent. in all tuberculous lesions in children, not only in mild and local lesions, but in severe and generalized ones.

Orth lays stress on the point that a bovine infection in childhood may be the source of a pulmonary phthisis in later life; new infection of the neighboring tissue can take place or infection in a distant organ (endogenous reinfection). This infection with the bovine bacillus, moreover, does not produce immunity against re-infection (exogenous reinfection).

L. Revillet[17] has observed that in certain locations in France tuberculosis is very rare among adults, but that many children die of tuberculous meningitis and peritonitis, or suffer from tuberculosis of the bones. He thinks infection from individual to individual is excluded and attributes the evil to ingestion of milk or meat infected with the bovine tubercle bacilli. He has found a high percentage of infected cattle in these regions.

C. Catteneo[19] reviews the literature on the identity of the human and bovine tubercle bacillus and reports his results in a series of tuberculous children with bovine and human tuberculosis.

He found that cases of surgical tuberculosis reacted more frequently to the bovine, while those of pulmonary tuberculosis reacted to the human type.

Nobécourt and Darre[21] examined the blood of fifty-one infants for tubercle bacilli. Thirty-five of the children suffered from tuberculosis, sixteen from non-tuberculous disease. Five to 20 c.c. of blood were obtained by venous puncture and injected into guinea-pigs. In none of the non-tuberculous infants were the bacilli found and in only four of the tuberculous. These observations coincide with those of other writers and show that, as in adults, the tubercle bacilli rarely occur in the blood.

M. P. Nobécourt[22] examined the urine of children by means of guinea-pig inoculation to ascertain the frequency of tubercle bacilli. Positive results were obtained with the urine of children afflicted with renal tuberculosis, subacute pulmonary tuberculosis, pulmonary tuberculosis with cavity formation and miliary tuberculosis. It was never found in cases of amyloid infiltration of the kidney, or those of hemorrhagic or chronic nephritis.

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L. Jeanneret,[23] in his study of the intradermal tuberculin reaction in children, applies the test in this way: The skin on the anterior surface of the forearm is lifted between the fingers and slowly, nearly parallel with the surface is injected .0001 c.c. of old tuberculin so that a small white area of edema results. The reaction appears at the end of twenty-four hours and reaches its height after forty-eight hours. The lesion produced is a papule circular, if the injection has been correctly made, bright red or rose colored; at times it is white and edematous. Around the papule is a halo of erythema. The size of the papule varies little, but the violence of the reaction is determined by the size of the halo. From the second or third day the lesion retrogrades. The peripheral halo disappears first, the papule becomes violet colored, then brownish, but it is usually two or three weeks before it completely disappears. The author subjected 900 children to this test and has drawn some interesting conclusions. He thinks it has advantages over the von

Pirquet test, finding it more sensitive, for in cases of acute disease, as measles, typhoid, whooping-cough and influenza, the von Pirquet becomes negative sooner and reappeared later than the intradermal test.

Another advantage is that the amount of tuberculin used can be accurately determined, as it cannot be with the von Pirquet test. A positive intradermal test reveals, according to Jeanneret, a tuberculous focus in the organism, active, torpid or cicatrized. In spite of this specificity it does not serve in diagnosing tuberculosis among adults, for all react positively, but it is an excellent test in children up to 7 years of age. Negative results are of great value and give almost absolute surety that the subject is not tuberculous. If the clinical examination reveals tuberculosis, a positive intradermal test also confirms the diagnosis. If the clinical examination is doubtful, a positive test is a strong argument for the presence of tuberculosis.

The author found that the violence of the reaction corresponded to the importance of the lesion and, if a series of tests were made in the same individual, important information regarding prognosis could be obtained. When the tuberculous process ameliorated, there was a progressive diminution in the size of the intradermal reaction. This was true in most of the cases of tracheobronchial adenopathy treated with tuberculin. On the other hand, when the patient's condition became aggravated the intradermal reaction increased, parallel with the rise of temperature and emaciation.

Jeanneret feels that the value of tuberculin treatment can be determined by the violence of the intradermal reaction. The treatment is not fit for patients in whom the first intradermal reaction is of large size, as this shows a hypersensibility to tuberculin. If the lesion is larger than 8 mm. in diameter tuberculin should not be administered.

According to A. Brown,[24] in hospital practice at least 90 per cent. of the children under 2 years who show a positive von Pirquet reaction are affected with an active and fatal tuberculosis, and with rare exceptions the tuberculous lesions are general in distribution. A negative cutaneous reaction in infancy, except when the infants are moribund or suffering from measles, is almost conclusive evidence against the existence of a tuberculosis, but in suspicious cases a second and even a third test should be made.

K. Dluski and S. Rudzki[25] report their results with Moro's tuberculin test. The object of their study was to ascertain whether the test substantiates the diagnosis when the physical examination speaks for the presence of a tuberculous lesion, and whether the test is of any prognostic value. To perform the test a piece of the salve about the size of a pea, made of 5 c.c. old tuberculin and 5 gm. lanolin, was rubbed into the epigastrium from a half to three quarters of a minute. A positive reac-

tion was denoted by the appearance of a rash consisting of numerous red nodular areas. Three grades of reaction were noted. Grade I: The presence of one to fifty nodules, limited to area of inunction, and not accompanied by vesicle formation. Grade II: Presence of more than fifty nodules and extending beyond the area of inunction. Grade III: More than fifty nodules with vesicle formation.

The authors made 579 tests on 500 tuberculous patients. Two hundred and eighty-eight patients, or 50 per cent., showed reactions of the first grade; seventy-one, or 12 per cent., of the second, and twenty-two, or 4 per cent., of the third grade. In 198 patients the reaction was negative.

The authors conclude that Moro's tuberculin reaction is a specific reaction, and its presence is proof of tuberculin infection. A negative reaction, however, does not exclude tuberculosis, because in 35 per cent. of the cases there was a negative reaction. The test is of more prognostic than diagnostic value. The absence of a Moro's reaction in a patient with clinical tuberculosis shows an unfavorable prognosis. The more marked the reaction, the better the prognosis. The test is particularly useful for control of the tuberculin treatment.

H. Rozenblat[27] does not believe that a single von Pirquet test excludes tuberculosis, for he has found certain tuberculous individuals to give a positive reaction after several negative ones. Fifty-four children with active and twenty-five with latent tuberculosis he treated simultaneously with .0001 mg., .001 mg., .01 mg. and 0.1 mg. of tuberculin in order to ascertain whether the quantitative test is of value in judging the stage or form of the disease, but his results merely confirmed the fact already known that active tuberculosis was more sensitive to tuberculin than inactive tuberculosis, and the form and stage could be differentiated to only a limited extent. Nor was he able to obtain more severe reactions by repeated injections.

He is of the opinion that the cutaneous reactions are to a certain degree dependent on the characteristics of the skin. He quotes a number of writers, who believe the cutaneous reaction to be merely local and to have nothing in common with a general tuberculous allergie. These authors support their view by the results obtained with other substances, as toxins of colon-typhus, dysentery bacilli and other bacteria. The author himself has often with 1 per cent. peptone solution obtained reactions identical with the tuberculin reactions. These facts seem to Rozenblat sufficient to put down the tuberculin test as non-specific.

F. L. Wachenheim[28] finds a much lower percentage (8.9 per cent.) of positive von Pirquet reactions among children over 6 years than do most observers. He thinks a positive von Pirquet should always be regarded seriously and the patient treated as if he were the victim of a tuberculous lesion.

V. C. Vaughan[30] is of the opinion that a method of testing the capability of the individual to produce an antituberculin ferment would materially add to any value that might be attributed to the tuberculin test. In his tests Vaughan used the conjunctival method. He instilled the tuberculin into the conjunctival sac, and in case of a negative reaction repeated the instillation in eight or nine days. A primary reaction he calls the reaction of infection. It consists of a hyperemia of the mucous membrane of the conjunctiva, of the lower lid and inner canthus. It is tardy in making its appearance, reaches a moderate degree of severity and subsides slowly. It is due to a reaction between the tuberculin and the antituberculin ferment present in the fluids of tuberculous subjects. The secondary reaction occurs as a result of the re-instillation. With the first instillation the cells of the conjunctiva develop the antituberculin ferment, and when the tuberculin is again dropped in the eye the ferment unites with it and produces the secondary reaction. This reaction is characterized by rapid development, high degree of inflammatory reaction and rapid subsidence.

Vaughan studied these reactions in normal children below 1 year of age, in individuals with advanced lesions, with early lesions and with healed lesions. He draws the following conclusions as to the value of the ophthalmic reaction: 1. A positive reaction following the first instillation of tuberculin into the conjunctival sac is strong confirmatory evidence of the existence of tuberculous disease. 2. A negative reaction following the first instillation of tuberculin is of no value in determining the presence or absence of tuberculosis. 3. A secondary reaction obtained in an eye previously unaffected by instillation is strong evidence against the presence of tuberculous disease, at least in an active form. 4. Failure to obtain a secondary reaction in an eye unaffected by previous instillation is of no value in determining the presence or absence of tuberculosis.

St. Engel[32] has carefully studied the anatomy and topography of the bronchial glands in children, and has worked out a method for the identification of normal and enlarged bronchial glands in Roentgen-ray plates. He thinks bronchial gland tuberculosis is a very important subject, as it is often an isolated process and the only manifestation of the tuberculous disease.

The bronchial glands are the lymph-nodes which accompany the air passages to the divisions of the large bronchi and those situated within the lungs. The glands which accompany the trachea and bronchi are divided into two groups; those in the right and left tracheobronchial angle, the tracheobronchial glands, and those in the angle of division into the two principal bronchi, the bifurcation glands. The pulmonary glands also make up two groups, the broncho-pulmonary glands which

lie outside the lung in the angle of division of the first collateral branch into the lateral branch, and the pulmonary glands which lie within the lung in the angle of division of the small bronchi. The first of these, together with the pulmonary glands in the neighborhood of the hilus, make up the group known as the hilus glands, and are the ones which play an especial rôle in the bronchal gland tuberculosis of children.

Roentgen-ray pictures of the tracheobronchial tree: The position of the trachea, bronchi and glands in Roentgen-ray plates is considered in relation to the median shadow. This is the shadow caused by the thymus (in infants), the heart, and blood-vessels. Hochsinger, describing this shadow in radiographs of children's chests, says that in young children never more than two arches are seen, though four occur in adults. The shadow has a bottle-like form with a broad body and small neck. The neck reaches from the first and second to the fifth or sixth thoracic vertebra. The neck represents the blood-vessels and thymus, the body the heart. In infants the heart shadow is wider and longer and the neck is shorter and wider than in older children. In older children the heart shadow is one or two vertebra lower and has three, sometimes four, arches (outward curves). The character of the median shadow does not always correspond to age; sometimes in older children it resembles that usually found in infants, and vice versa. The trachea and principal bronchi in childhood lie entirely within the median shadow. The left principal bronchus reaches much nearer the clear pulmonary field than the right. The bronchi, which are a direct continuation of the principal bronchi, run nearly parallel with the borders of the cardiac shadow. Usually, and, especially in very young children, the right one makes a brighter streak near the right auricle, and after giving off the first collateral branch, turns outward and then downward, with a slight curve, still running parallel with the heart shadow, but outside its borders. In children with a high, wide shadow, however, it remains within the shadow. On the left side the bronchus, which is a continuation of the principal bronchus, always remains within the ventricular shadow and runs parallel with its border.

The pulmonary hilus in Roentgen-ray pictures: The right pulmonary hilus lies at the edge or just outside the median shadow. The left is covered by the cardiac shadow. The left lies deeper than the right. In young children the right lies at the level of the junction of the fifth rib with the spine, the left at the level of the sixth. In older children both hili lie at the level of one rib lower.

The topographical relation of normal bronchial glands: The glands located in the angle of bifurcation always lie within the median shadow. The right tracheobronchial glands lie within the median shadow, very near the border, or just outside the border. The left tracheobronchial

glands lie further from the edge and within the shadow of the spinal column. The right bronchopulmonary glands in young children lie at the edge of the median shadow within the pulmonary field and at a level with the junction of the upper and lower cardiac arches. The left bronchopulmonary glands lie within the median shadow at the upper end of the ventricular arch.

Tuberculous bronchial glands in Roentgen-ray pictures: The only conclusion justifiable is that a shadow in radiograph corresponds to an enlarged bronchial gland, when it corresponds topographically to the gland. There are many sources of error. The shadows are often indefinite and room for guess work is so great that only autopsy examination can control the findings. Suspicious shadows can, however, be studied as to form, size and exact topological relations to the bronchial tree, so that some conclusions can be drawn. There is a typical picture of tuberculous bronchopulmonary glands of the right side. This group, as described above, lies in the right pulmonary field and has a characteristic form due to the position in the angle of division of the bronchi. If the bronchial tree is not visible these glands can be recognized through being separated from the cardiac shadow by a bright line. The left hilus glands, on the other hand, are not so easy of identification because their shadow falls within the cardiac shadow.

The bifurcation glands are quite invisible, even if considerably enlarged. This is to be regretted since they are the glands oftenest enlarged and caseated and sometimes the only ones affected.

From this study the author concludes that the region of the bronchial glands usually falls within the area of the median shadow, the farther within, the younger the child; only the right hilus lies in the clear pulmonary field or near enough to it for the glands to be recognized. The Roentgen-ray picture of tuberculous bronchial glands is then, on the whole, unsatisfactory, and from it positive diagnosis can rarely be made.

To his former report of twenty-one cases of hilus tuberculosis studied with the Roentgen-ray, E. Sluka[83] adds seventeen, and the findings substantiate those on the first series. The shadow indication of hilus tuberculosis as described by Sluka in his first article is of triangular shape with its base superimposed on the median (heart and blood-vessels) shadow and with the apex reaching into the pulmonary field. It is more frequent on the right than on the left side. The author found, however, that the shadow was not always triangular. On the right side three different forms were observed, namely, the triangular shadow, a semicircular shadow with the convexity upward, and a semicircular shadow with the convexity downward. On the left side only two forms were observed, a shadow, reaching from behind the heart toward the periphery, semicircular in shape with the convexity either upward or downward.

The author explains the shadow as a pulmonary infiltration around the hilus glands, not as the primary pulmonary lesion or the pleuritic exudate, which so frequently accompanies the primary lesion. He never found the shadow during the first two years, as he undoubtedly would have done were it due to a pleuritic exudate, which usually occurs at this time of life.

Sluka feels that little progress has been made in the clinical diagnosis of hilus tuberculosis in children. He obtains more information from percussion of the anterior than posterior parts of the chest. Dulness on the left side above the heart, though often physiological, is suggestive, as is also dulness on the right near the sternum and over the right middle lobe.

From clinical observations and Roentgen-ray studies Sluka concludes that hilus tuberculosis in children can easily be overlooked, and that in the early stage it can be recognized only by Roentgen-rays.

Shortly before their death, E. Rach[34] took Roentgen-ray pictures of six children suffering from tuberculosis and compared the findings with autopsy findings. He also took Roentgen-ray pictures of the chests of 783 healthy school children. He was able in these two series of radiographs to demonstrate shadows which correspond to Ghon's primary lesion, to lesions at the hilus, and to pleuritic exudates. He says a Ghon's primary lesion can be diagnosed in children with negative physical findings, but with a positive von Pirquet test, by a bean-shaped, sharply-defined shadow isolated and free in the pulmonary field. In many of these cases the history of a possible infection can be obtained. The question of prognosis is important, when a Ghon's lesion is demonstrated, and, according to most authorities, this is better the older the affected child. It is unfavorable if the lesion occurs in nurslings, but after the age of 8 the prognosis is good, as the lesion is usually a stationary one. The presence of Ghon's lesions explains most satisfactorily the positive von Pirquet test in these children.

Two of the author's pictures showed a triangular shadow with the base superimposed on the median shadow and the apex extending into the pulmonary field. In both instances these corresponded to caseated lymph-nodes at the hilus. In cases in which the paratracheal glands were involved, the radiograph showed a shadow which ran in an upward curve from the median shadow, then parallel with the trachea, and finally returned again to the median shadow.

Rach believes that a pleuritic exudate is indicative of a primary pulmonary lesion, and this exudate can be demonstrated in Roentgen-ray pictures. In two of his pictures it was seen as a sharp linear shadow, corresponding to the interlobular fissure. The findings were confirmed by autopsy.

E. Rach[35] reports the following: A child became ill with indefinite symptoms, loss of appetite, headache, occasional vomiting and slight rise of temperature. Examination of the eye backgrounds revealed a choked disk. The tuberculin reaction was faintly positive and resembled a cachectic reaction. A radiograph of the chest showed a typical picture of miliary tuberculosis. Scattered through both lungs were diffuse shadows the size of hemp seed.

The child died soon afterward of general miliary tuberculosis.

G. Simon[36] has been able to demonstrate the primary pulmonary lesion in radiographs. He says that in cases of bronchial gland tuberculosis radiographs show within the pulmonary area sharply defined shadows. They stand in topological relation to the hilus glands and evidently represent an older lesion than the lesion in the glands. Moreover, they are not in a situation where lymph-glands are usually located and must be regarded as a pulmonary lesion.

One of the author's cases was especially interesting. A ten-year-old boy had a chronic diffuse bronchitis. The von Pirquet test was positive. Roentgen-ray picture showed tuberculous bronchial glands of both sides. Also far to the right in the upper lobe were two small shadows in a location where pulmonary glands are hardly looked for. There was little doubt that they were tuberculous lesions and the origin of the hilus tuberculosis, for from them delicate lines of shadow radiated toward the hilus glands and were interpreted as a peribronchitis in the course of the lymph vessels.

L. Rebodeau-Dumas and A. Weil[37] believe that with proper technic the diagnosis of miliary tuberculosis can be made with the Roentgen-ray. In three cases they found in radiographs small round spots, varying in size and clearness, scattered through both lungs, in the apices as well as at the bases. Enlargement of the tracheobronchial glands could be demonstrated in the plates. Autopsy proved the shadows to correspond to miliary tubercles.

A fourth case was particularly interesting as the Roentgen-ray picture was the means of diagnosis. The patient was in a typhoid state and gave a positive tuberculin test. On auscultation only disseminated bronchial râles could be heard. A radiograph showed a number of small shadows similar to those in the other plates and a diagnosis of miliary tuberculosis was made.

The authors call attention to the fact that one must be careful not to mistake the shadows of blood-vessels, bronchi or glands scattered through the parenchyma of the lung for miliary tubercles.

I. O. Woodruff[38] studied the Roentgen-ray findings in the chest of fifteen children with tuberculous meningitis. He found no evidence of tuberculosis in one, tuberculous glands and some involvement of right

lower lobe in two; more or less extensive involvement of one or both lungs in twelve. Of six patients who died he compared the Roentgen-ray findings with the autopsy findings and observed that they agreed. He therefore concludes that the Roentgen-ray pictures can be relied on for diagnosing tuberculous areas of the bronchial glands and lungs. He concludes, moreover, that, since autopsy records show a pulmonary tuberculosis beyond incipiency in 88 per cent. of the cases of tuberculous meningitis, a routine radiographic examination of the chest might be of real value in determining the diagnosis, especially in those cases in which the von Pirquet test is negative, and it is impossible to demonstrate tubercle bacilli in the spinal fluid.

B. M. Wronker[39] reports a number of instances of papulo-necrotic tuberculid in infants and discusses their significance. These lesions are described as small, round, slightly raised, rather hard, flat papules, ranging in size from a pinhead to a millet seed; in color, red, reddish brown, livid; often presenting on their surface a small dry crust, which is easily removed and leaves a whitish, central depression with occasionally a bleeding point in the center. If put on a stretch, they have a glistening appearance. The lesions generally appear in crops and heal spontaneously in from four to eight weeks.

The author observed instances of papulo-necrotic tuberculids in infants from 3 to 11 months old, and this finding led to the diagnosis. In all, it was corroborated by findings in the lungs, the von Pirquet test, or by the autopsy examination. Generally, there were few lesions (one to nine), but in one case they were too numerous to count. In six of the cases the lesions were situated on the lower extremities; in one on the chest and back; in one on the back and upper extremity, and in one they were scattered over face, back, chest and thighs. They conformed closely to the description given.

The writer is of the opinion that these lesions are indicative, in the majority of cases, of general miliary tuberculosis, and hence are of ominous prognostic significance.

E. W. Fiske[40] has made a routine examination of the blood in children suffering from tuberculosis of the bones and osteomyelitis to determine whether this procedure facilitates the differentiation. He found that in two-thirds of the cases the average white-blood count during the acute stage of the disease was 11,600 in tuberculosis, and 16,200 in osteomyelitis, a difference of 5,000 leukocytes. That this difference is a constant and reliable factor for diagnosis is shown by the fact that in osteomyelitis but 24 per cent. of the counts were below 12,000, as compared with 66 per cent. in tuberculosis, while 39 per cent. of the former were over 18,000, and but 13 per cent. of the latter were 18,000.

Leukocytosis occurs not when the bone infection is due to the tubercle bacillus, but only when it is caused by a pyogenic organism. The only

cases difficult to diagnose from a white count alone are those of osteomyelitis with a normal count or slight leukocytosis, as happens oftener than most text-books affirm. In these, the relation of the count to other symptoms, such as fluctuating count with temperature, freely draining bone abscess, or a long-standing, low grade process in a case in which the count has at one time been high, would point to a diagnosis of osteomyelitis.

SYMPTOMATOLOGY

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Rohmer[41] differentiates the forms of tuberculosis as they occur at different periods of childhood. During the first year there is a primary focus in the lung and involvement of the corresponding bronchial glands. This is followed by a generalized tuberculosis. From the second to the sixth year there are more cases of tuberculous meningitis than of generalized tuberculosis. At this time also occur glandular and osseous tuberculosis, miliary tuberculosis and caseous pneumonia. During the school age the forms found in the adult predominate, but the mortality and morbidity are lower than at any time during childhood.

J. Cassel[42] reports the clinical and autopsy findings in six cases of tuberculosis in infants, and points out as of greatest value the tempera-

ture charts, which were kept over a long period of time—in three children during their whole life. A study of these charts shows that the temperature was at first but slightly above the normal, or normal with an occasional rise, but that finally it always took on a remittent character with a difference of two or three degrees (Centigrade) between morning and evening. The length of this period of remittent temperature varied from three to twelve weeks and lasted until the child's death. Cassel thinks the temperature is of especial value for diagnosis, if the cutaneous test is negative and there are few or no findings in the chest. It is due either to the development of a new crop of tuberculous lesions or to a mixed infection with streptococci or staphylococci.

The weight curves also were of interest. There was always a drop in weight, corresponding to the early rise in the temperature. If the temperature dropped to normal, the weight curve remained stationary; when it developed its remittant character, the weight curve dropped rapidly.

F. Hamburger[43] states that tuberculosis in the adult can be understood only through a knowledge of tuberculosis in childhood. He again compares the course of the disease with that of syphilis. The similarity consists not only in there being in tuberculosis as in syphilis a primary lesion with regionary lymph-nodes enlargement, immunity against reinfection and a tendency to recurrence, but also a primary, secondary and tertiary stage in both.

The source of infection is undoubtedly a patient with phthisis, and it may be necessary for the child to come in contact with the infected individual only once to become infected himself. The question as to portal of entrance cannot be answered with absolute certainty. Pathological anatomists have found that in 95 per cent. of the children who have a tuberculous lesion the primary focus is in the lungs, and the secondary one in the bronchial lymph-glands. In the other 5 per cent. the primary lesion is in the skin, intestine or genitals. Often tuberculous conditions among children are recurrent. The organism is unable to render the bacilli innocuous and the disease becomes manifest.

The first symptoms of a primary tuberculous infection in childhood are fever, emaciation and cough, but these are frequently overlooked, and symptoms of the recurrence are the first to which attention is paid. Spina ventosa, spondylitis, coxitis, phlyctenular conjunctivitis, serous pleuritis, peritonitis and pericarditis are manifestations of recurrences of the tuberculous process.

Tuberculosis of the cervical lymph-nodes is usually the awakening of an infection which occurred years before. Sputum containing tubercle bacilli comes in contact with the tonsils. The bacilli are carried into the glands and remain latent as long as the organism is strong. Tuber-

culous cervical adenitis frequently occurs after the acute infectious diseases, especially measles.

The prognosis depends on whether the disease is primary or recurrent, and on the age of the child. Eighty per cent. of the children infected before they are a year old die, while only 10 to 20 per cent. of those infected after two years die.

In the method of treatment Hamburger lays more stress on general measures, believing that the tuberculin treatment is still in the experimental stage, and that more children have been harmed than helped by it.

A. L. Goodmann[45] reports an unusual case of rupture of a mediastinal lymph-node into the bronchus. The child, a boy of 5 years, was admitted to the hospital because of painful urination. The urine showed many leukocytes, few blood cells and bacilli, which on culture proved to be colon bacilli. Cystoscopy revealed on the right side near the trigone an ulceration which was not thought to be tuberculous. The child improved under small doses of hexamethylenamin. On the eleventh day the nurse found the child in bed, cyanotic and breathing heavily. The pupils were dilated the head thrown back, its arms drawn up, the legs spastic. A tentative diagnosis of tuberculous meningitis was made. A few hours later the child went into collapse. Artificial respiration, intubation and tracheotomy were in vain. An insufflation apparatus was connected with the end of the catheter, and, as the end of the catheter was moved back and forth, a thick, cheesy mass exuded from the tracheotomy wound. On the expulsion of this mass the child began to breathe more freely, the lips reddened and cyanosis disappeared. He did not regain consciousness for four or five hours. The next day a Roentgen-ray picture was taken and there were found shadows indicative of enlarged bronchial glands. This finding, associated with the bladder condition, assured the author that he was dealing with a broken-down lymph-node, which had ruptured into the bronchus, or was producing asphyxia by constricting the trachea.

Goodman reviews the literature of similar cases and discusses the subject of enlarged bronchial glands. He says that without roentgenoscopy it is in the great majority of cases impossible to make a diagnosis. The symptoms may be divided into those showing involvement of the respiratory, the circulatory or the digestive apparatus, or a combination of all three. Symptoms of the respiratory type are slight respiratory sounds on auscultation, cough and more or less labored breathing. Of the circulatory type, cyanosis; of the digestive, fever, vomiting, followed by dyspnea and cyanosis. All cases are not of tuberculous origin, and in the case reported tubercle bacilli could not be demonstrated.

H. Koch[46] has studied the conditions favoring the development of tuberculous meningitis. He found that the time of year had a marked

influence, and that 61 per cent. of fatalities occurred from January to June, with the maximum number in April. Certain years of life showed a decided predisposition. Sixty-eight per cent. of all cases were found in the first four years, but the second year showed the largest absolute number of cases. Infection from tuberculous parents occurred in but 23 per cent. of the cases, and there appears to be no family predisposition to tuberculous meningitis, since in only one family did a number of sisters and brothers die of the disease. Boys and girls were affected equally. Breast feeding was no safeguard against the disease — in fact, 82 per cent. were breast-fed and eighteen artificially-fed children.

Previous attacks of measles seemed to play an important rôle. One hundred and twenty-nine of the 305 children had had measles, and sixty within the year that meningitis developed. In ten instances a study of the clinical and autopsy findings showed a direct relationship between measles and meningitis. Of the other infectious diseases only whooping-cough seemed connected with tuberculous meningitis. In thirteen cases there was a history of trauma, but in two only did the autopsy point to a connection between the two.

Clinically, manifest tuberculous disease occurred in forty-nine cases, but in nearly all careful autopsy examination showed a primary lesion in the lungs, with regional lymph-node involvement. In no case was there a primary intestinal lesion. In 250 cases the pulmonary lesion was still active, and the writer does not believe a tuberculous meningitis can develop from a calcified lesion.

R. L. Pitfield[48] reports in detail the cases of three children who had tuberculous meningitis, but recovered. One was that of Rumples, discussed at the medical society of Hamburg, when he showed the brain of a boy who died of phthisis. When 9 years old this child had had an attack of meningitis with the following symptoms: retraction of the neck, hyperesthesia, absence of knee-jerk and bilateral optic neuritis. Lumbar puncture yielded a fluid containing tubercle bacilli. After repeated lumbar punctures the boy recovered. For some months he was dull and stupid, but eventually regained his normal mental condition. He died of phthisis eight years after the attack. At autopsy the membranes over the fissure of Sylvius on either side were thickened and glued together, but miliary tubercles were no longer present.

From his study the author concludes that, perhaps, one of two hundred children with tuberculous meningitis recovers; steps should be taken to realize this possibility. The patient should be put in a quiet dark room. Lumbar puncture should immediately be performed and frequently repeated. Forced feeding of milk and eggs should be given through a nasal tube. Morphin should be administered for the pain and hexamethylenamin freely given. One or two injections of tuberculin are advised.

A. Rongel[51] does not believe that there is an antagonistic action between asthma and tuberculosis as some writers hold. He performed a von Pirquet test on one hundred and one asthmatic children and obtained positive reactions in fifty.

O. Frankfurter[52] declares there is a causal relation between asthma and tuberculosis, and thinks that a systematic tuberculin cure will cause amelioration and finally a cessation of the attacks.

A. Lediard[53] reports an unusual case of chyliform effusion, due to obstruction of the receptaculum chyli by tuberculous glands. The patient was a boy of 18 years. When a child he suffered from attacks of severe abdominal pain which were diagnosed as tuberculous peritonitis. When first seen by the writer, he was very emaciated, the abdomen was distended with a fluid which passed down the left inguinal canal into the scrotum when he coughed. The distention of the abdomen caused rapid respiration and cardiac displacement, and on this account operation was advised. An incision was made above the umbilicus and six pints of milky fluid were obtained. The patient died two weeks later with symptoms of tuberculous peritonitis.

Autopsy revealed a wide-spread tuberculous process. The peritoneum, omentum, mesenteric glands, liver, spleen, kidneys, lungs and brain all showed a deposit of tubercles. The receptaculum was apparently obliterated in a mass of glands and fibrous tissue, and the lymphatics were all distended. The ascites was the result of a transudation, since no perforation was found.

P. Reyher,[54] in a previous contribution, gave as his opinion that there exists a relationship between orthostatic albuminuria and tuberculosis. A study of twenty cases of orthostatic albuminuria substantiates this opinion. In the twenty patients he found more or less pronounced symptoms of bronchial gland tuberculosis. In one-half the number there was an hereditary taint; in 25 per cent. there were well-marked symptoms of scrofula; all of these patients had an elevation of temperature; and in a number the Roentgen-ray picture showed the presence of enlarged bronchial glands. The tuberculin test gave the most important evidence. It was positive in all patients, and as nine of them were children below 10 years, this is significant.

The author believes that toxic substances circulating in the blood for a long time cause the albuminuria.

J. Oraison[56] in the course of five years has observed three instances of renal tuberculosis in little girls. Two of the children had tuberculous family histories, and one child suffered from Pott's disease. The question whether the kidney is primarily or secondarily affected has not been settled.

The clinical picture in children does not differ from that in adults except for the addition of one almost invariable symptom, incontinence of urine. In all cases of tuberculosis of the kidney the functional power of the organ supposed to be healthy should be ascertained. This can be accomplished only by catheterization of the ureters, which is possible even among young children and quite easy after six or seven years.

The treatment for children is the same as for adults, except that medicinal treatment may be given a little longer trial, as all the renal tissue is needed to combat the infections so numerous in childhood.

Nephrectomy is the operation advised.

H. L. Rocher and J. Ferron[57] report observations on renal tuberculosis in children. For diagnosis of the affected side they used the endoscope and ureteroscope. In one instance a child of 11 years had suffered for three months with frequent urination and pain at the end of urination. One month before admittance to the hospital there had been a few drops of blood in the urine. The cystoscope showed the mucous membrane of the bladder reddened and ulceration around the trigone on the left side. The right ureteral orifice was normal, the left appeared as a black spot through which a sound was easily passed. Ten centimeters of urine was obtained from this side, which contained pus-cells and tubercle bacilli. A few days later catheterization of the right side gave a normal urine. The left kidney was removed and found to be the seat of tuberculous cavities and granulomata.

As in adults, nephrectomy is indicated in renal tuberculosis of children, but only when the integrity of the other kidney is established. This, the writers affirm, can easily be done by means of the ureteral catheter. The ureter of a child of 5 admits a No. 40 tube, 7 cm. long.

E. Moro[58] does not agree with R. Pollack that erythema nodosum is a tuberculous lesion. Moro, who studied thirty cases — four of which were absolutely negative to any tuberculin test — does not wish to say, however, that there is absolutely no relation between the two conditions. In two instances he saw erythema nodosum, associated with purpura, develop after the inunction with Moro salve. This seems to point toward a connection between erythema nodosum and purpura, and purpura very frequently accompanies tuberculosis. There are only two ways to determine absolutely whether the two conditions are related. The first is to find the tubercle bacilli in the lesion and the second to produce a local reaction in the lesion after the injection of tuberculin.

PROGNOSIS

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G. Tugendreich,[59] says that the discovery of the von Pirquet test has modified views concerning the prognosis of tuberculosis in infants. Infants showing a positive von Pirquet test are always tuberculous, but can live beyond the first year, although that was not formerly thought possible.

Tugendreich has been able to follow the fate of ten infants who gave a positive test during the first year of life. Of these, nine died; five in the first year, two in the second, one in the third, one in the fourth. One child is living and is four years old. The children who died during the first year all succumbed to the tuberculous process, while the others died of an intercurrent infectious disease.

The author looks into the questions, Do these children suffer more from infectious diseases than non-tuberculous children, and is the death-rate higher among them? From the study of a small number of statistics, his own and those of medical literature, he does not get the impression that tuberculous disease causes an increased susceptibility to measles and scarlet fever or an increased mortality from them, but that from diphtheria the mortality is greater in tuberculous children under 2 than in non-tuberculous children. His statistics for the mortality of tuberculous infants coincides with those of Schick and Pollack.

M. Hollensen[60] says until recently it was believed that a positive von Pirquet in a child under 2 years meant active tuberculosis and an utterly unfavorable prognosis. She has, however, treated with tuberculin a number of infants showing a positive von Pirquet test and feels that the treatment was not without results. At first she gave rather large doses, rapidly increasing them, according to the Schlossman method, but later found small doses were more effective. She began with 0.0000001 to 0.000001 grams and increased 1/10 to 2/10 of the dose every two or three days, if there was no reaction. On a rise in temperature, the dose was decreased to the point of producing no temperature. The cases were divided for study into three groups. Group I included twelve children between six weeks and one year. Of these only two died of tuberculosis, six were cured, two improved but died of an intercurrent disease, and two were still under treatment

at the time of writing. Group II included thirty-four children from 1 to 2 years. Twenty-four, or 70.6 per cent., took the course of treatment; nine, or 26.5 per cent., were cured; six, or 17.6 per cent., died of tuberculosis. Group III included nine children over 2 years. Seven showed good results, two showed no benefit.

Discussing the significance of a negative von Pirquet test after a course of tuberculin treatment, the writer concludes that the negative result is due to a true immunity against tuberculin. This opinion seems to be substantiated by the fact that those children who were cured by the course of treatment and who showed a negative von Pirquet at the termination of treatment still showed a negative von Pirquet test two to three years later and remained well, while those who were not cured had again a positive von Pirquet test and signs of tuberculosis.

P. Lederer[61] says two facts show that the prognosis of tuberculosis in infants is not absolutely unfavorable. The first is the result of autopsy examinations. In older children lesions in the curative stage have been found and in infants who have died of pulmonary tuberculosis areas with incipient reparative process show that the organism of the child is not absolutely at the mercy of the virus. Then pulmonary tuberculosis and tuberculosis of the other organs, as skin, bones and joints, must be considered separately, for the latter has a much more favorable prognosis.

Of his own experience with pulmonary tuberculosis, the author publishes the histories of three cases in infants who have long since passed the first year. Tubercle bacilli were not found in the sputum but the diagnosis was based on the physical findings, the positive tuberculin reaction and the Roentgen ray pictures. The author feels that he confirms the statements of Pollack and others who say that tuberculosis in infants is not always fatal, as it was formerly thought, and he agrees with Rietchel that, when the child does recover, the number of infecting bacilli or their virulence is not so great as to overpower the regional lymph-nodes.

L. Lawatschek,[62] studying the prognosis of tuberculosis in infancy, has attempted to ascertain the longevity of infants giving a positive von Pirquet test. Until recently such cases were regarded as hopeless, but lately it has been shown that with careful regulation of food and hygiene and the systematic use of tuberculin the outlook is not so bad. In congenital tuberculosis and tuberculosis acquired during the first quarter of a year, naturally the mortality figures run highest. In four cases of placental tuberculosis the children were watched from birth and three died of tuberculosis between three and six months; the other was well and did not show any sign of the disease up to nine

months. Since the discovery of the von Pirquet reaction the writer has vaccinated every infant whose mother had active tuberculosis. Of seventy-seven infants seventeen gave positive reactions. The youngest of these were 4, 8, 10 and 11 weeks. The first child remained only two days with the mother and had no tuberculous manifestations during the eight weeks of observation. At nine months he developed a bronchitis and was then lost sight of. The other three died in three or four months of phthisis and meningitis. Of eleven children born of tuberculous mothers and giving a positive von Pirquet test, seven died before the sixth month. The author also vaccinated in his ambulatory clinic all children of tuberculous parents. In this group the youngest child giving a positive test was thirty-eight days old. The infection was an acquired one, with clinical manifestations. At the time of writing the child has reached the third year and is in good health.

Osseous tuberculosis, which is rare in infants, has a relatively good prognosis, accounting for the fact that the child survives in spite of the early infection. Besides the age of the child and the localization of the lesion, the severity of the infection and the opportunity for reinfection influences the prognosis. Rietchel has shown that the prognosis depends on the number of infecting bacilli. Children die more rapidly from an extra-uterine repeated infection than from a congenital infection, if they are protected from reinfection.

R. Cruchet[63] discusses the prognosis of subacute tuberculosis in infants. Its gravity is conceded by all authors, but statistics differ as to the fate of children who give a positive von Pirquet during the first year. Hutinal and Tixier found that death usually resulted in the month after a positive test. Of ten children observed by Calmette, one died of convulsions a month after the positive von Pirquet, but the other nine were in good health seven to eight months later. Thus one can conclude that tuberculous infection among infants may be cured; not only cutaneous and glandular forms, but also pulmonary, intestinal and even meningeal tuberculosis. In the last ten years reports on remissions and even cures of tuberculous meningitis have multiplied to such an extent as to leave no doubt that they do occur. Yet the author says that, though the cure of tuberculosis is possible in infancy, it is less frequent than among children above 2 years and adults. He finds the explanation in the fact that there is a tendency to generalization of infection which is due to a special morbidity in infancy, and this is independent of the mode of infection.

C. P. Lapage and W. Mair,[65] from the study of the post-mortem records of the Manchester Children's Hospital, conclude that among children the mortality from tuberculosis rises during the first year, reaches its maximum in the second year and then falls rapidly. Glandu-

lar tuberculosis is very common and in a fair proportion of cases the disease is limited to the glands. In the Manchester Hospital the abdominal glands were more often affected than the thoracic, but most commonly both sets were affected. Tuberculosis of the thoracic and abdominal glands was frequent without any accompanying lesion of the lung or intestine. Apical tuberculosis is not rare, even among infants (in eleven of the cases apical lesions were found), and the form designated as chronic phthisis, though not very common, does occur in young children. Chronic cavitation is also found even in young children, but usually there is then a tendency for the disease to spread in the lungs and to become generalized. Tuberculous meningitis most often follows generalized or miliary tuberculosis but it not uncommonly occurs as a complication of glandular or bony tuberculous lesions.

W. Weinberg[66] has studied the records of 5,000 tuberculous families and followed the history of the children (18,000 in number) up to the twentieth year, noting the mortality and fecundity percentage among them. He found that the number of children born to the tuberculous was much lower than to the non-tuberculous, especially in families where the husband was the victim.

The mortality during the twenty years was 47 per cent., against 40 per cent. among children of non-tuberculous parents. This included specific and general mortality. The nearer the birth of the children was to the death of the parents, the higher was the mortality, so that children born during the parents' last year showed a death rate of 90 per cent. of the total death rate for the twenty years. The mortality was influenced by social position and alcoholism, being higher among the lower classes. The mortality was higher among the children who lived with their infected parents, but not higher than among those living with other tuberculous individuals. The mortality was increased by diseases of the respiratory tract and whooping-cough. Measles, scarlet fever and diphtheria did not influence the mortality. On the whole the statistics emphasize the danger of direct contagion. Proofs of constitutional influences were few.

Peters[67] obtained the history of 19,000 schoolchildren, in order to ascertain how many had an hereditary tuberculous taint. He found that 531, or 5.6 per cent., had parents, brothers or sisters with tuberculous lesions, while in 356, or 3.7 per cent., the grandparents, uncles or aunts were infected. Physical examination showed that the children in Group II were better developed than in Group I.

Peters found also that of 470 children, whose parents, brothers or sisters had tuberculosis, 337, or 71.5 per cent., gave a positive, and 133, or 28.3 per cent., gave a negative von Pirquet test, while of 108

whose grandparents, uncles or aunts were infected, 58, or 53.7 per cent., gave a positive, and 50, or 46.3 per cent. a negative reaction.

These statistics speak for the probability of direct house infection in many cases in which there is an hereditary taint.

PROPHYLAXIS

68. Cozzolino, O.: Maternal Nursing by Tuberculous Mothers, Arch. f. Kinderh., 1913, lx-lxi, p. 221.

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71. Van Pelt, J. V.: The Architecture of Open Air Schools, Med. Rec., New York, 1913, lxxxiv, 20, p. 880.

72. Knopf, A.: Rest and Exercise for the Tuberculous and Predisposed School Child, Med. Rec., New York, lxxxiv, 20, p. 875.

73. Bacon, C. S.: Sanatorium Treatment of Tuberculous Gravidae, Puerperae and Their Children, Ill. Med. Jour., 1913, xxiii, 141.

O. Cozzolino[68] discusses under the following heads the question whether a tuberculous mother should nurse her baby. Does the milk of a tuberculous woman contain tubercle bacilli? Does it contain toxins? What influence has nursing on the mother and how does it affect the baby?

As it has been proved that only rarely are living tubercle bacilli eliminated by a healthy mammary gland and even if eliminated are not virulent, such infection, according to Cozzolino, can generally be ruled out. Animal experiments have shown that, without doubt, the tubercle toxins can be eliminated in the milk and give rise to general (cachexia) and local manifestations (chronic gastro-enteritis). The farther advanced the process, the greater is the amount of toxin eliminated. The presence of the toxin in the milk of mothers in the early stage of tuberculosis has been demonstrated in this way: Tuberculous children, or those in whom tuberculosis was suspected, were tested for the Moro reaction with the milk of tuberculous women mixed with vaselin. The writer, however, feels that further research must be made on the human before it is proved that in the early stages of the disease sufficient toxin can be eliminated in the mother's milk to produce cachexia and marasmus. Neither must it be disregarded, he feels, as an eliminating fluid for immune bodies, which increase the resistance of the breast-fed infant, but he does not agree with Schlossman that breast milk is in itself a protection against tuberculosis or can favorably influence a tuberculous process.

As regards the effect on the mother, it is deleterious, and because of the increased anemia, the milk supply runs so low she is obliged to desist; a manifest tuberculosis usually becomes accentuated and a latent process active. Disregarding all this Cozzolino thinks a tuber-

culous mother should not nurse her infant because there is danger of contact infection. He is surprised to find so many writers vacillating and undecided, when all rules of prophylaxis point to separation of the child from the tuberculous mother. He says that, even if these children develop well during the nursing period, they all give a positive von Pirquet and therefore have a latent tuberculosis. According to the teaching of von Behring, an infection acquired in infancy can remain latent through the entire period of childhood and awaken during adolescence and puberty.

Cozzolino finds it impossible for a mother to nurse her child and carry out all proper precautions unless she is especially trained in bacteriologic and antiseptic methods; even then there is always danger of an error in technic.

This interesting paper is concluded with the remark that the problem merges itself into that of general prophylaxis of tuberculosis in childhood.

J. W. Brannan[69] discusses the subject of open-air schools. He divides them into three groups: Those for children with pulmonary tuberculosis; those for children who are anemic, under weight, ill-nourished and hence predisposed to tuberculosis; and those for normal children.

The open-air school movement began in 1904 with the *Waldschule* in Charlottenberg. In January, 1908, it spread to the United States and with the opening of the next school term there will be 200 fresh air schools, according to the statement of the National Association for the Study and Prevention of Tuberculosis.

The fresh air schools vary greatly in their construction and arrangement but in régime they are practically the same—life in the open air, nourishing food, rest in bed after dinner and systematic gentle exercises. The results are practically alike in all—gain in weight, disappearance of the signs in the chest and improvement in the general condition, physical and mental.

The results in the fresh air schools of the second group, those for anemic children, also are excellent.

The third group, for normal children, is in some respects the most interesting and full of promise. That the public and parents in particular are beginning to appreciate the value for the children of fresh air during school hours is shown by the fact that they are preparing to demand it of the school authorities.

A. G. Rice[70] pleads for the open window school, rather than the open-air school. The open-air school was instituted with impulsive enthusiasm and consequences other than hygienic were not looked to. Even on the hygienic side it has been disappointing and has not proved to be the desired panacea.

The author emphasizes that the improved condition of children of the open window school favorably compare with that of children of the open-air schools. From the economic and educational points of view the open window schoolrooms excel the open-air schools. The cost of maintaining the open-air schools is greater than the original outlay. It is almost impossible to furnish efficient instruction in them, since they must be conducted as ungraded schools. From the moral viewpoint the open-air school is objectionable. It tends to develop the child asymmetrically and, by emphasizing physical training, leads to a consciousness of bodily defects. The child, seeing that at some one's else expense he is fed, clothed and given time to sleep, becomes slowly to regard himself as a special case, to feel in the end that the world owes him a living.

TREATMENT

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75. Beck, C.: Treatment of Tuberculosis in Children with Rosenbach's Tuberculin, Ztschr. f. Kinderh, 1913, v-vi, p. 438.

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90. Pybus, F. C.: Remarks on Tubercular Cervical Adenitis, Clin. Jour., 1913, xlii, 24, p. 370.

91. Müller, G. P.: Treatment of Tubercular Cervical Adenitis, Ann. Surg., 1913, lviii, No. 4.

A. Czerny[74] has inquired into what factors influence the course of tuberculosis among children. It was once thought that increased body weight could prevent the spread of tuberculous disease and favorably affect the course, but it has been observed that the best nutritional condition in children has been followed by a tuberculous infection of no mild course. On the other hand, children with a low body-weight and a proved presence of a tuberculous lesion have gone on for years without the disease making rapid progress.

Hereditary taint does not explain the difference in the course of the disease. The author has had opportunity to watch, in families free from tuberculosis, children who became infected from an outside source and in whom the disease was rapidly fatal. In tuberculous families he has seen children in whom the disease was mild and did not end fatally. Of all the factors thought to influence the disease only one seems worthy of mention. Tuberculosis has a better prognosis in children with stable nervous systems. The reason is difficult to explain. Excitable nervous children are difficult to nourish because of their changing moods, but a lowered state of nutrition, as mentioned before, does not explain this matter. It is more likely due to a rapidly changing blood supply, to the various organs dependent on vasomotor disturbances so frequently found in these nervous children. An excitable, nervous system is congenital but can be modified by proper conditions and it is always advisable to send nervous children with tuberculosis to sanitariums, away from home. Sanitariums, Czerny finds, should be located where the climate is not extremely variable. Rapid changes of temperature and high winds favor the development of respiratory diseases and these have a bad influence on the tuberculous process. In most instances, however, respiratory diseases of children seem to arise independent of climatic conditions and are due to a constitutional abnormality, called the exudative diathesis, which can be completely cured by the regulation of diet. Eggs and milk must be avoided and vegetables and cereals freely given.

Sanitariums for tuberculous patients must be fully equipped for heliotherapy and fresh air treatment. Fresh air in the writer's estimation is more important than sunlight. Also such institutions should be in a position to induce pneumothorax in cases of pulmonary tuberculosis. This procedure, according to Vogt and Pielstieker, can be used for very young children and in Czerny's opinion is a great step in advance.

As to tuberculin the author has not yet seen results which could not have been obtained without its use. Czerny lays stress on the influence of the diet on the course of the disease. He so regulates the food that there is no rapid water retention. A rapid gain in weight

usually means water retention, which from animal experiments seems to cause a more rapid progress of the disease. He advises a diet as follows: Twice daily, meat, vegetables and malt coffee, and a minimum amount of milk. Cod-liver oil is also given. The children observed on this diet remained for months at a stationary weight, but they grew, their muscles became hard and none developed new tuberculous areas during the treatment.

C. Beck[75] treated a number of children suffering from various forms of tuberculosis with Rosenbach's tuberculin. Rosenbach produces his tuberculin by planting a fungus on a six to eight weeks old culture of tubercle bacillus, developing it at 20 to 22 C. for ten to twelve days, separating the culture mass from the medium, and mixing it with a glycerin-carbolic acid solution. Experiments on animals showed this tuberculin to be much less toxic than Koch's old tuberculin.

Beck's observations were made on cases of pulmonary tuberculosis, exudative pleuritis, peritonitis, tuberculosis of the glands, bones, soft parts, ears, intestines and skin. A number showed multiple lesions. The injections were given as follows: The first dose was either 0.1 or .01 grams, and the dose was increased 1/10 gram every second day until 1 gram was given; then this dose was given twice a week for a month, then once a week for a month, so that the course of treatment usually lasted three months. In obstinate cases the treatment was continued half a year or longer.

General reactions occasionally occurred after injections. After large doses a few of the children complained of headache, loss of appetite, nausea, slight temperature. Marked subjective symptoms were never observed. The local reactions were frequent and severe. A few hours after the injection redness and swelling appeared at the site and spread over the entire arm. In twelve to twenty-four hours the process resembled erysipelas or phlegmon, but subsided quickly on the application of moist compresses. The so-called "focal-reaction" was constant. In cases of pulmonary tuberculosis the râles increased in the affected area. They disappeared again but reappeared with each new injection, although to a slighter and slighter extent; the fluid in cases of pleurisy and peritonitis increased; lymph-nodes grew more swollen at first but gradually became smaller and finally disappeared. Tuberculous abscesses opened, eliminated pus, and finally healed, tuberculous ulcers threw off the necrotic tissue and healed. On the whole, the results with Rosenbach's tuberculin were favorable, but, as Rosenbach says, the injections must be repeated to maintain the immunity produced by the use of the tuberculin.

In his ambulatory clinic, W. Wolf[76] treated with tuberculin twenty-two children suffering from scrofula and incipient tuberculosis. He

wished to ascertain whether this method is practical. He usually began with 1/10,000 mg. and gradually increased the dose, giving one injection a week until 1/1,000 mg. was given, and then further increased the dose to 1/100 mg. If untoward effects developed he continued with this dose and omitted one week before continuing. In four cases he began with a larger dose (2/10,000 mg.) but reduced it because of the reaction. This method differs from Wittich's, who began with 1/1,000 mg. and gave two injections a week.

The most interesting results were obtained in relation to the von Pirquet test, which was influenced in nearly every case, but disappeared in only three. Clinically, improvement followed in every instance. There was retrogression of the glandular swellings, the rhinitis, and the ocular inflammations. Subjective symptoms, loss of appetite, weariness, pain in the chest were mitigated soon after the second or third injection. These results corresponded closely with Wittich's, yet Wolf asks whether the same could not have been obtained with less dangerous methods. Scrofulous and tuberculous children react very favorably to dietetic and hygienic measures, and the author feels that at the present time the tuberculin treatment should not be carried out in an ambulatory clinic. He does not think that the necessary precautions to prevent harmful effects of the tuberculin can be taken.

H. Koeppe[78] noticed that frequently after a von Pirquet test, axillary glands, which had been swollen, rapidly decreased in size, but only on the side on which the test was made. He concludes that the tuberculin entered the lymphatics, was carried to the glands, and there exerted a curative effect. The following of his cases was the most striking: The child was 4½ years old, emaciated and cachectic. The chest was flat but showed no signs of tuberculosis. In the right axilla was a mass of glands, as well as along the edge of the right sternomastoid muscle. The von Pirquet test made on the right forearm was strongly positive. After the test the child unexpectedly improved generally, gained in weight and the glands became much smaller. A second von Pirquet test four months after the first resulted in a still greater reduction of the glandular swellings. Koeppe believes this was due to the action of the tuberculin which was directly carried to the glands by the lymphatics. As there was still considerable enlargement of the cervical glands, a third von Pirquet test was made behind the ear, since the lymphatics from this region empty into the cervical glands. These glands also decreased in size. Four years later the child was perfectly well.

Other patients showed similar improvement and Koeppe hopes that an easy and effective method of treating tuberculous glands will develop from this study.

C. A. Julian[80] reports his own observations on the children treated by von Ruck with the von Ruck vaccine, as well as on 131 children treated by himself.

Biological and experimental studies by von Ruck demonstrated the amount of specific protective substances in the blood serum before and after the vaccination and showed that they developed in response to the administrataion of the vaccine. After its administrataion there was an increase in the specific agglutination, the opsonic index and the specific amboceptors. The bactericidal power of the blood serum of children treated with the vaccine was apparent from the fact that guinea-pigs inoculated with tubercle bacilli and exposed to the action of the serum taken before vaccination died of tuberculosis, while those inoculated with the same number and same culture of tubercle bacilli, survived when the tubercle bacilli were exposed to the action of the serum taken after vaccination. Regarding the technic of preparing the vaccine, Julian refers to von Ruck's original paper.

Symptoms suggestive of tuberculosis, including cough and rise in temperature, disappeared during the first four months after vaccination. All the children gained in weight and strength, had better color, and were more interested in their studies. Sixty-four of the children were examined fourteen months after the vaccination. In those who had inactive tuberculosis the findings were practically unchanged. In those who had active lesions there was a decided amelioration and retrogression of the physical signs. In all but one case a decrease in the size and number of the lymph-nodes was noted.

Three weeks after vaccination the 131 new children showed as remarkable a change as those of the first group. There was gain in weight, retrogression of physical signs, and disappearance of enlarged glands.

R. C. Cummings[81] experimented with von Ruck's tuberculo-protein. One cubic centimeter of the vaccine was mixed with 2 c.c. of sterile normal saline containing 0.25 per cent. phenol. Of this solution 0.30 centimeters were injected for each of ten successive weeks into ten apparently healthy guinea-pigs. Five weeks after the course of treatment they were inoculated intraperitoneally with 0.001 to 0.1 mg. of virulent tubercle bacilli. At the same time nine control animals were injected intraperitoneally with the same amount of virulent tubercle bacilli. About fourteen days later the vaccinated animals seemed sick and at the end of twenty-one days were obviously so. At the end of four weeks the controls appeared ill in quite the same way. All died of general miliary tuberculosis, the average length of life being the same in both series. Cummings concludes from these experiments

that immunity was not produced by the vaccine, which seemed rather to have enhanced susceptibility than otherwise.

Ten children with active tuberculous lesions were treated by L. C. Ager[82] with mixed vaccines. He says that, although recent investigators have shown that tuberculosis alone may produce the tissue changes in the lungs commonly found in phthisis, it is believed that such changes are usually produced by a secondary invasion of various pyogenic organisms. The vaccine Ager used contained the bacillus of influenza, the bacillus of Friedländer, the *Micrococcus catarrhalis,* the pneumococcus, the streptococcus, the staphylococcus and diphtheroid bacilli. This product was chosen because it contained the organisms found in washed specimens of sputum. The results were disappointing and no definite conclusions could be drawn. Autogenous vaccines it was thought might have shown a different reaction.

L. Emmett Holt, discussing this, said he believes that such reports should not be made and such therapy is harmful, for the unwary may be tempted to pin faith on a specific, and to neglect the routine method of treatment.

Galliot[83] has had opportunity to treat a number of children suffering from tuberculosis by the remineralization method of Ferrier. Each day for several months he gave two or three of the following tablets: Carbonate of calcium, phosphate of calcium, each 20 to 30 centigrams; chlorid of calcium, 10 to 20 centigrams; calcined magnesia, 5 to 10 centigrams. He gave cod-liver oil also.

He does not believe that complete cicatrization of the lesions was obtained, or the future of the patients assured, but in all cases he found the disease to be arrested. Most remarkable was the amelioration of general symptoms and the gain in weight. Galliot recommends this treatment for the children of the poor who can not be moved to other surroundings.

O. Vulpius[84] emphasizes the importance of sanatoriums for the treatment of bone and joint tuberculosis in children. Such institutions should be in a healthful location in the country near enough to the city to be accessible by the surgeon. They should be thoroughly equipped for orthopedic and surgical operations, with balconies, solariums for fresh air and direct sunlight, and laboratories for physical, physiological and bacteriological examinations.

L. Revillet[86] describes the method of treating surgical tuberculosis on the Mediterranean coast, and the results obtained.

The patients each day are given a sea bath for one to three minutes and then are exposed to the direct rays of the sun, at first for from one-half to one hour, later from two to three hours, then for the greater part of the day. Large hats are worn to prevent headaches and, when

the heat is very intense, white gauze is placed over the exposed surface to prevent solar erythema. The marked pigmentation of the skin is a favorable sign. Immobilization is employed only for acute painful inflammations of joints and surgical intervention as little as possible.

The author says that the improvement in the children under the influence of the sea air, sunlight and baths is surprising. Not only is there a transformation of the general condition, but local lesions heal rapidly; swellings and suppurations—glandular, articular and osseous—disappear; cutaneous lesions cicatrize and the scars are remarkable for their firmness, suppleness and white color.

A. H. Tubby[88] gives four reasons for improvement in the treatment of surgical tuberculosis among children: (1) The wide recognition of the beneficial effects of climatic treatment; (2) the employment of Roentgen rays in diagnosis; (3) the wiser administration of tuberculin; (4) the better education of surgeons in the use of supporting apparatus and greater ingenuity in inventing new apparatus.

Outlining the leading principles of a scheme for dealing with surgical tuberculosis, the author says that every case in the country should be registered, that hospitals for the treatment of these children in a suitable climate should be organized and supervised by medical men who have received special training. Children should be admitted without unnecessary routine and the length of their stay should be determined by the nature of their disease. All children should be followed up after dismissal and suitable trades sought for tuberculous cripples. Moreover, special provision must be made for incurable cases, so they will not be a source of infection. There must be opportunity also for research work in surgical tuberculosis.

J. H. Jopson[89] discusses the fallacies in the treatment of tubercular adenitis. He says that there is a tendency on the part of some surgeons to recede from surgical treatment and to substitute conservative measures. The simple incision and curettement of broken down glands in conjunction with hygienic measures was recommended by some, and Roentgen ray, tuberculin therapy and Bier's hyperemic treatment by others. It seems to Jopson that the extravagant claims of the upholders of the Roentgen ray treatment are unwarranted, or founded on insufficient data, and that, when physicians condemn the results of operative treatment, they ignore the work of men like Dowd, Wohlgemuth and von Noorden, who have shown that cure followed radical treatment in from 70 to 90 per cent. of the cases. The Roentgen ray is useful as an adjuvant to surgical treatment, while tuberculin therapy is protracted, expensive and contraindicated where there are caseation, liquefaction and necrosis present. In these cases Jopson also recommends the removal of enlarged and diseased tonsils and adenoids. He has

frequently seen them removed before the operation for the glands, but only exceptionally was the operation on the glands thereby avoided.

F. C. Pybus[90] says that tuberculous cervical adenitis is almost always secondary. The chief sources of infection are pediculi capitis, suppurative inflammation of the middle ear resulting from tuberculous infection of the petrous bone, carious teeth, tuberculous skin lesions, and, most important, infection from adenoids and tonsils. The supraclavicular nodes may become infected from the mediastinal, or both groups may be a part of a generalized infection. A tuberculous tonsil may be enlarged but more commonly it is small, hidden and likely to be overlooked. The enucleation of the tonsil is essential if it is the infecting focus. When this is done the glands may undergo a natural cure, but if there is caseation the glands should be removed. Operations are difficult and should be undertaken by only a skilled surgeon. There may be occurrences even after dissection and the author emphasizes the necessity of general sanatorium treatment. He has yet to see that tuberculin exercises much influence on tuberculous glands.

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